Ontario [Commissions and committees of inquiry] Committee appointed to report upon the pollution of air, soil, and water in the townships of Dunn, Moulton, ...
Report. 1968

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Report, 1968

REPORT ON POLLUTION

ONTARIO

REPORT

OF THE COMMITTEE

APPOINTED TO

INQUIRE INTO AND REPORT UPON THE

POLLUTION

OF AIR, SOIL, AND WATER

IN THE TOWNSHIPS OF

DUNN, MOULTON, AND SHERBROOKE

HALDIMAND COUNTY

SEPTEMBER, 1968

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COMMISSIONERS

GEORGE EDWARD HALL
M.D., Ph.D., D.Sc., F.R.S.C.
Chairman

W. C. WINEGARD
B.Sc., M.Sc., Ph.D.

ALEX McKINNEY

Counsel to the Committee
RODGER A. GORDON, LL.B.

Secretary to the Committee
MAX E. WEISSENGRUBER, B.A.

To: The Honourable,
The Minister of Health
of the
Province of Ontario.

Mr. Minister:

We, the undersigned, appointed by Order-in-Council dated the 6th day of November, 1967, as amended by Order-in-Council dated the 11th day of January, 1968, respectfully submit to you our report upon the matters referred to us by the said Orders-in-Council.

Alex McKinney
[illegible]
G. Hall

Dated at Toronto
this 25th day of
September, 1968.

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[Seal]

ONTARIO

PROVINCE OF ONTARIO

ELIZABETH THE SECOND, by the Grace of God of the United Kingdom, Canada and Her other Realms and Territories Queen, Head of the Commonwealth, Defender of the Faith.

TO George Edward Hall, Esquire, of the Town of Orillia,
Chalmers Jack MacKenzie, Esquire, of the Village of Rockcliffe, and
Alex McKinney, Esquire, of the Township of Chinguacousy.

GREETING:

WHEREAS in and by Chapter 323 of The Revised Statutes of Ontario, 1960, entitled "The Public Inquiries Act," it is enacted that whenever Our Lieutenant Governor in Council deems it expedient to cause inquiry to be made concerning any matter connected with or affecting the good government of Ontario or the conduct of any part of the public business thereof or of the administration of justice therein and such inquiry is not regulated by any special law, he may, by Commission appoint one or more persons to conduct such inquiry and may confer the power of summoning any person and requiring him to give evidence on oath and to produce such documents and things as the Commissioner or Commissioners deems requisite for the full investigation of the matters into which he or they are appointed to examine;

AND WHEREAS Our Lieutenant Governor in Council of Our Province of Ontario deems it expedient to cause inquiry to be made concerning the matters hereinafter mentioned;

NOW KNOW Ye that We, having and reposing full trust and confidence in you the said George Edward Hall, Chalmers Jack MacKenzie and Alex McKinney DO HEREBY APPOINT you to

be our Commissioners and naming you, the said George Edward Hall to be the Chairman thereof,

to inquire into and report upon the pollution of air, soil and water in the Townships of Dunn, Moulton, and Sherbrooke in the County of Haldimand and its effect upon human health, livestock, agricultural and horticultural crops, soil productivity and economic factors within the said area;

AND WE DO HEREBY CONFER on you, Our said Commissioners, the power to summon any person and require him to give evidence on oath and to produce such documents and things as you Our said Commissioners deem requisite for the full investigation of the matters into which you are appointed to examine;

AND WE DO HEREBY FURTHER ORDER that all Our departments, boards, commissions, agencies and committees shall assist you, Our said Commissioners, to the fullest extent, and that in order to carry out your duties and functions, you shall have the authority to engage such counsel, research and other staff and technical advisers as you deem proper;

TO HAVE, HOLD AND ENJOY the said Office and authority of Commissioners for and during the pleasure of Our Lieutenant Governor in Council for Our Province of Ontario.

IN TESTIMONY WHEREOF We have caused these Our Letters to be made Patent, and the Great Seal of Our Province of Ontario to be hereunto affixed.

WITNESS: THE HONOURABLE WILLIAM EARL ROWE, A Member of Our Privy Council for Canada, Doctor of Laws, Doctor of Social Science,

LIEUTENANT GOVERNOR OF OUR PROVINCE OF ONTARIO at Our City of Toronto in Our said Province, this sixth day of November in the year of Our Lord one thousand nine hundred and sixty-seven and in the sixteenth year of Our Reign.

BY COMMAND

(Signed) ROBERT WELCH,
PROVINCIAL SECRETARY.

ORDER-IN-COUNCIL

Arising out of the original grant of Letters Patent, it was necessary to obtain a further Order-in-Council as one of the Commissioners so appointed was unable to act.

Copy of an Order-in-Council approved by His Honour the Lieutenant Governor, dated the 11th day of January, A.D. 1968.

The Committee of Council have had under consideration the report of the Honourable the Minister of Health, dated January 9th, 1968, wherein he states that,

WHEREAS by Order-in-Council numbered OC-4729/67 dated the 6th day of November, 1967, made under The Public Inquiries Act, your Honour did appoint the Commissioners mentioned in the said Order to inquire into and report upon pollution of air, soil and water in the Townships of the County of Haldimand, mentioned in the Order, and the other purposes mentioned therein;

AND WHEREAS Dr. C. J. MacKenzie, one of the Commissioners named in the said Order has resigned;

The Honourable the Minister of Health therefore recommends that Dr. W. C. Winegard, B.Sc., M.Sc., Ph.D., President of the University of Guelph, be appointed a Commissioner in the place and stead of Dr. C. J. MacKenzie, resigned.

The Committee of Council concur in the recommendation of the Honourable the Minister of Health and advise that the same be acted on.

Certified,

(Signed) J. J. YOUNG,
Clerk, Executive Council.

PREFACE

Statement of Sequence of Events
to be followed by the Committee

On November 6th, 1967, The Province of Ontario, through an *Order-in-Council,* established a Commission "to inquire into and report upon the pollution of air, soil and water in the Townships of Dunn, Moulton and Sherbrooke in the County of Haldimand and its effect upon human health, livestock, agricultural and horticultural crops, soil productivity and economic factors within the said area".

The Commission was issued pursuant to *The Public Inquiries Act,* R.S.O. 1960, Chapter 323, appointing G. Edward Hall as Chairman. Under the Act, the Commissioners "have the power of summoning any person and requiring him to give evidence on oath and to produce such documents and things as the Commissioners deem requisite to the full examination of matters into which they are appointed to examine."

Early in October, 1967, public allegations were made concerning the possibility of air, soil and water pollution in the area. Prior to this date, several government Departments, including the Department of Health, had been investigating the various agricultural, industrial and health problems in the area. After weeks of preliminary planning and careful consideration of the problem, the Committee was established to investigate, in detail, the complete range of air, soil and water pollution problems in the designated area.

The Commission will investigate the situation by calling witnesses, by commissioning recognized specialists to undertake specific studies, by examining the world's relevant scientific literature, by conducting comparative analyses of the health of people and of animals, acreages under crops, numbers of animals, production, quality, prices, etc., over a 10 to 15 year period. Inquiry will be made into the practices, controls, waste products of local industries. The Commission is concerned with discovering the actual situation so that the report may be factual and therefore of value.

Although the Commission may deviate from its preliminary plans as evidence and accumulated knowledge warrants, it has re-

quested specific studies by various specialists in the following areas of concern:

1. A study of epidemiological, pharmacological and toxicological factors of relevant industrial pollutants as they specifically relate to humans.

2. A clinical sciences study of the diagnostic and therapeutic aspects of relevant industrial pollutants as related to humans and human well-being.

3. A veterinary sciences study of the physiological, pharmacological, toxicological, biochemical, pathological and clinical considerations of relevant industrial pollutants as related to:
   (a) Cattle—beef—re health, weight gains or losses, residuals in meat, quality, etc. Cattle—dairy—health, weight gains or losses, milk production quality, etc.
   (b) Poultry—health, weight gains or losses, egg production, quality.
   (c) Hogs, sheep, etc.—re above where applicable.

4. A study of the effects of relevant industrial pollutants as related to:
   (a) Cereal crops—re yield, quality, etc.
   (b) Forage crops—re yield, quality, etc.
   (c) Vegetable crops—re yield, quality, etc.

5. A study comparable to 4 (above) but as related to:
   (a) Fruit crops—e.g. apples, peaches, berries, re yield, quality, etc.
   (b) General vegetables—e.g. grass, coniferous trees, deciduous trees, shrubs, flowering plants, etc.

6. A study of the effects of relevant industrial pollutants on soil, as related to:
   (a) Alterations of physical and chemical properties.
   (b) Soil nutrients.
   (c) Productivity.
   (d) Soil analysis—a comparative study.

7. Although the influence of relevant industrial pollutants on the nutritive value of all products, beef, milk, eggs, vegetables, fruit, etc., would be considered in the several specific studies, it is considered advisable to institute a study on the general nutritional considerations.

8. A study of the economic aspects of relevant industrial pollutants as related to:
   (a) Land and property values—a comparative study.

(b) Influences on materials, e.g. paint, wood, masonry, metal, glass, etc.
(c) Production—a comparative study.
(d) Quality—a comparative study.
(e) Prices of produce and land—a comparative study.
(f) Acreage under various types of crops—a comparative study.
(g) Animal populations on the different farms—a comparative study.

9. A study of the meteorological factors which may be significant, e.g.
   (a) The collection of meteorological data over a 10-15 year period covering prevalent winds, precipitation, fogs, temperatures, etc.

10. A study of the industrial practices in local industries and in similar industries:
    (a) In other parts of Canada, U.S.A., U.K., Europe, etc.
    (b) The methods of control of industrial pollutants in these plants and their effectiveness.
    (c) Health hazards in these plants and their control.

11. The Commission has requested that a paper be prepared on the chemistry of the "halide" group of chemicals and on other chemicals which are found in relevant industrial pollutants.

Most of the above studies will take the form of reviews of the world's scientific and industrial literature. In such literature will be found the results of much research conducted in many different parts of the world; such literature has recorded the experiences of others engaged in work comparable to that of the Commission; the literature also contains opinions and facts, assumptions and guesses, substantiated records and unproven contentions, all of which must be digested and sorted to extract relevant, verifiable data.

It will be the task of the Commission to weigh the evidence, to obtain the facts, to consider the entire matter in a responsible manner and to report the result of their deliberations to the Minister of Health along with any recommendations considered by the Commission. At the same time the Commissioners wish it to be understood that the disposition of their report is strictly a responsibility of the Government.

The sequence of events, as envisaged by the Commission, is as follows:

1. The commissioning of the several studies (as suggested above) by selected independent consultants and specialists.

2. The calling of briefs from interested parties.
3. Public hearings to be held in the Cayuga County Court House in order that interested individuals, groups or associations may present evidence to the Commission and where individuals may be called to give evidence, present records or to be present for examination.
4. Study by Commission of briefs, evidence and exhibits.
5. Request any additional studies, research or analysis required.
6. Additional hearings in Toronto for the presentations by consultants and specialists, further opportunity for individuals or groups from the stated area to be heard on specific points, etc., the calling of other witnesses as required.
7. Preparation of the Commissioner's report.

It should be emphasized that the hearings and all other pertinent matters are under the complete and sole control of the Commission and its Counsel and that evidence which is presented is given under oath.

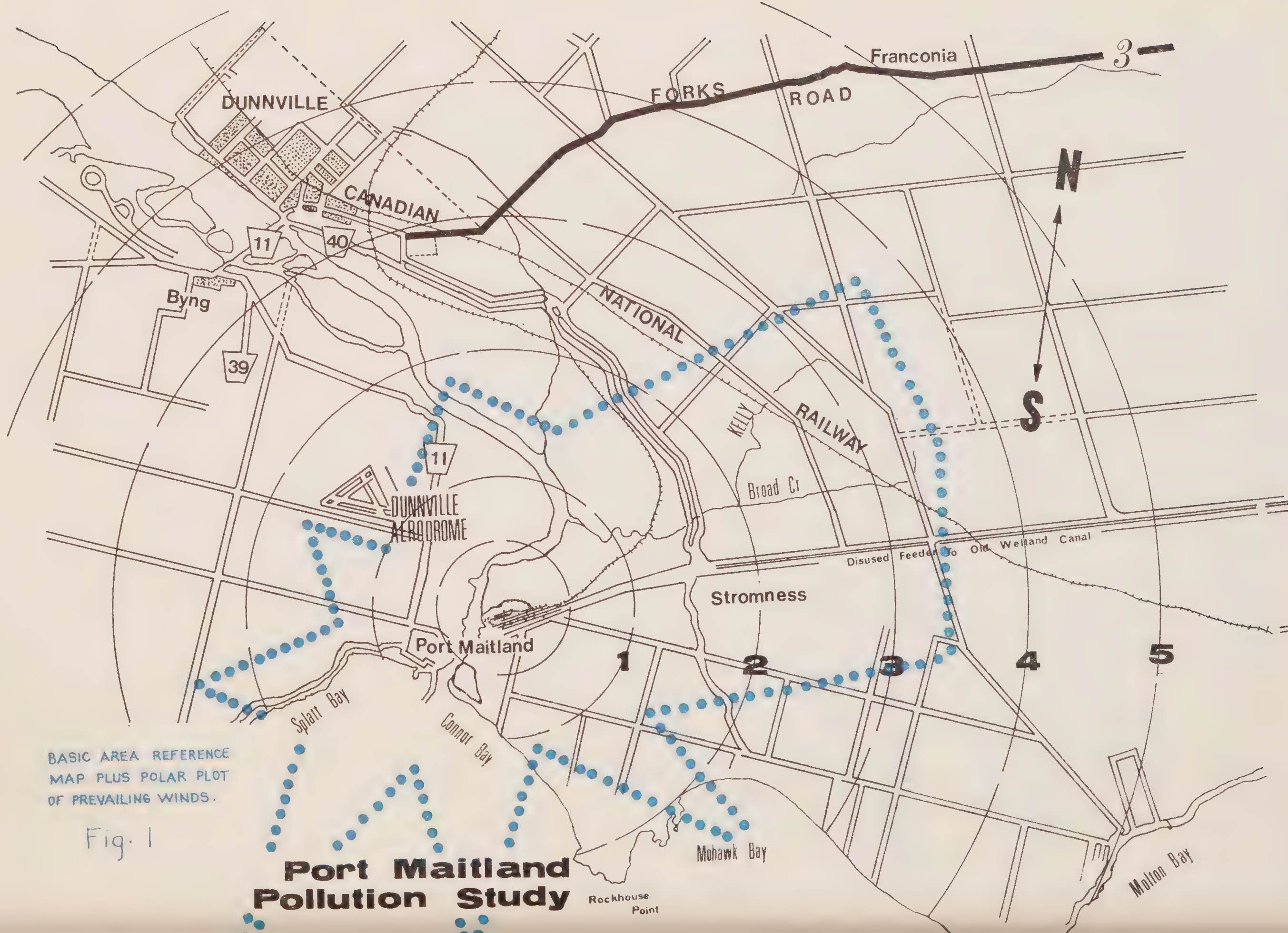
The Counsel for the Committee is Mr. R. A. Gordon of St. Catharines.

The Administrative Secretary is Mr. M. Weissengruber.

People wishing to contact the Committee should write to:

Mr. M. Weissengruber,
Secretary, Pollution Inquiry Committee,
Second Floor,
1 St. Clair Avenue West,
Toronto 7, Ontario.

Report of the Committee



BASIC AREA REFERENCE MAP PLUS POLAR PLOT OF PREVAILING WINDS.

Fig. 1

CHAPTER I

Introduction

A. THE ESTABLISHMENT OF THE COMMITTEE

1. By *Order-in-Council,* number OC-4729/67, dated November 6th, 1967, as amended by *Order-in-Council,* number OC-0172/68, dated January 11th, 1968, both approved pursuant to the provisions of the *Public Inquiries Act,* R.S.O. 1960, Chapter 323, we were appointed Commissioners "to inquire into and report upon the pollution of air, soil and water in the Townships of Dunn, Moulton, and Sherbrooke in the County of Haldimand and its effect upon human health, livestock, agricultural and horticultural crops, soil productivity and economic factors within the said area." (See *Proclamation.*)

2. We, the Commissioners, were accorded "the power to summon any person and require him to give evidence on oath and to produce such documents and things as you, Our said Commissioners, deem requisite for the full investigation of the matters into which you are appointed to examine"; and it was further ordered "that all Our departments, boards, commissions, agencies and committees shall assist you, Our said Commissioners, to the fullest extent, and that in order to carry out your duties and functions, you shall have the authority to engage such counsel, research and other staff and technical advisers as you deem proper."

3. Mr. Max Weissengruber acted as the Secretary of the Committee throughout its tenure. On November 17, 1967, Mr. Rodger Gordon, Barrister-at-Law, St. Catharines, was appointed Counsel to the Committee and acted in that capacity throughout the hearings.

B. THE GENERAL NATURE OF THE PROBLEM

4. The area under study consists of the Townships of Dunn, Moulton, and Sherbrooke in the County of Haldimand. The location of the Sherbrooke Metallurgical Company plant and the Electric Reduction Company plant relative to these three townships may best be ascertained by reference to the map of the area (Figure 1). It is important to note that the Town of Dunnville lies approximately four miles NNW of the two plants in question which are situated at Port Maitland.

5. Distances from the two plants, which are within 300 yards of each other, are indicated by circles of ½ mile, 1 mile, 2 miles, 3 miles, 4 miles, and 5 miles. The original large scale map, prepared at the request of the Committee, included township and county roads, concession and lot lines, and the names of the owners of the several parcels of land in the area (Figure 1).

6. Haldimand County, itself, lies in the southeasterly portion of South Western Ontario and is bordered on the south by the north shore of Lake Erie, on the west by portions of Norfolk and Brant Counties, on the north and NE by Lincoln and Wentworth Counties and on the east by Welland County. The County seat is the town of Cayuga.

7. The general and specific information on the meteorological or climatic factors which are of importance to this study were presented by our Consultant, Dr. D. Murray Brown, of the Department of Soil Science, University of Guelph. An attempt is made here to outline the manner in which pollutants are distributed by atmospheric conditions and the effect of such conditions on crops. Two meteorological factors are important when considering these problems:

(a) The ability of the atmosphere to transport and dispense pollutants;

(b) The effect of atmospheric conditions (both polluted and non-polluted) on crops.

8. In considering the first factor, it must be recognized that wind velocity and wind direction are the important influences in the movement of pollutants from original source to specific areas; e.g., light winds of a prevailing SW nature would mean that a concentration of pollutants might occur close to the original source in an area within the boundaries of a NE direction from the source. High winds, variable in direction, might mean that the pollutants would be dispersed rather evenly over a wide area.

9. Temperature, moisture, and sunshine are all important to the full development of plant life and may affect the ability of the plant to resist effects of air pollutants.

10. In order to study the effect of the above factors in the Port Maitland area, it was necessary to gather weather information over a considerable period of time so that a picture could be compiled of weather factors in that area.

11. The Federal Department of Transport maintains weather stations of several types throughout the entire country. Two types of stations are significant in this study—Principal and Ordinary:

(a) Principal reporting stations report on the hour, every hour for 24 hours a day. They report wind speed and direction at a height of 33 feet, temperature and precipitation at a 5 foot height as well as cloud cover, visability, distance, fog, station pressure, etc.;

(b) Ordinary reporting stations report temperatures, high and low for the day, and amounts and kinds of precipitation.

Prior to 1962, the Principal reporting station for the area was at Clear Creek, 25 miles SW of Simcoe on Lake Erie. After 1962, the Principal station was located at Simcoe, 10 miles inland from Lake Erie and 15 miles NW from Port Maitland. An Ordinary reporting station has been sited at Dunnville at the OWRC pumping station since March, 1962.

12. Wind direction is important in the movement of all pollutants from original sources. A study of wind directions for daytime and nighttime indicated that during the growing season, May through September, winds came in from a SW direction 24 per cent of the time. In addition, winds from the west and south together comprise 25 per cent of the total "wind directions." If winds from the west and south as well as SW are considered together, over 50 per cent of the winds come from this general direction. "Wind directions" during daytime hours (1 p.m. in the afternoon in July) occur 47 per cent of the time from the SW. When west and south wind directions are compiled with the SW figures, over 50 per cent of the winds blow in this general direction.

13. In the Port Maitland area, therefore, winds are predominantly from the SW. This is caused by the different "heating" characteristics of land and water surfaces. In the daytime, on sunny days, the land surface heats much more quickly than the water and, as a result, the air tends to move from the lake over the land and creates a "lake breeze" which is southwesterly in

direction. The situation changes at night (1 a.m.) when winds blow from the southwest only 16 per cent of the time. At night, when temperatures are lower, land surfaces are "cooling" and they set up a flow of winds from the land to the lake (15-20 per cent from the W, 15 per cent from the N and NE). Of interest also is the fact that the average velocity or wind speed at Malton, the nearest recording station for such data, is as follows:

0-4 m.p.h. — 16 per cent

4-12 m.p.h. — 63 per cent

Over 12 m.p.h. — 20 per cent

14. More than 80 per cent of wind speeds exceed 4 m.p.h., indicating a wider dispersion of pollutants than would be true if winds averaged less than 4 m.p.h.

15. Land areas near large bodies of water usually have higher humidities during daylight hours, due to the lake-breeze effect, than at night. During June, July, August (1955-60) at 1 p.m., the relative humidity averaged 12 per cent higher at Clear Creek than at London, but only 8 per cent higher at 7 o'clock in the evening. Little difference existed between the two sites at 1 a.m. These differences may be explained by contrasting the higher humidities caused by lake breezes, which affect Clear Creek, with the lower humidities in a more inland area—London—even though London is only 20 miles from Lake Erie.

16. The polar plot of the frequencies of wind direction at Port Maitland is of great significance to this study (Figure 1). This, together with the data on wind velocities, shows that the winds are chiefly from the S and SW and that wind velocities are in excess of 4 miles per hour 80 per cent of the time. Thus, it might be anticipated that smoke and/or fumes and/or dust from either the ERCO plant or the Sherbrooke Metallurgical plant, or both, would contaminate, particularly, that area lying to the N and NE of the Companies' plants, and to a lesser extent other areas near the plants.

17. Briefly stated, the question of air pollutants and their possible effects on crops and soil in the area was first considered in November, 1960. The Air Pollution Control Service, Ontario Department of Health, set out "limed" candles and "lead peroxide" candles in an area extending to a 5-mile radius beyond the industrial complex in Port Maitland. The candles were used to detect the presence of "fluoride" and "sulphur dioxide" in the atmosphere.

There was at that time no damage observed or claimed, but the Department of Health wished to be continually aware of the amounts of these two potential pollutants in the area.

18. The fore-runner of the ERCO plant was the Dominion Fertilizer plant which was built in 1958 and designed for the production of single phosphate fertilizer. In 1959, this company was purchased by ERCO and the plant was converted for the production of triple as well as single superphosphate fertilizer. Since phosphoric acid, rather than sulphuric acid, is used for the manufacture of the triple superphosphate, a new wet process phosphoric acid plant had to be built. This new plant began operation in July, 1961. However, as far back as July 20, 1959, a meeting was held by representatives of the Department of Labour, the Electric Reduction Company, and Mr. H. Nelson, Senior Engineer of the then Industrial Hygiene Branch of the Department of Health. The plans for the new plant, as mentioned above, were discussed.

19. It should be pointed out that there are two major manufacturing plants at Port Maitland, which is located on the Grand River just above its junction with Lake Erie. The ERCO plant uses, as basic raw materials, calcium phosphate rock which is brought in from Florida and sulphuric acid which is manufactured by the Sherbrooke Metallurgical Company Limited in its plant situated some 300 yards from the ERCO plant. The Sherbrooke Metallurgical Company produces, as well, zinc oxide, and in the process some sulphur dioxide and zinc compounds are emitted into the atmosphere. Therefore, at least two major potential pollutants, fluoride and sulphur dioxide, required specific measures for their control. Details of the industrial processes and the emission control practices of the two plants are presented in Chapter II.

20. It is significant to mention at this point that the Sherbrooke Metallurgical Company plant, being classified as a "metallurgical" processing plant, came under the standards and inspection of the Department of Mines, while the Electric Reduction Company plant came under the standards set by the Department of Health. This peculiar situation was corrected by the *Air Pollution Control Act, 1967,* which gave the Ontario Department of Health the responsibility for:

(a) The investigation of air pollution problems.

(b) Conducting research into the field of air pollution.

(c) Ensuring that industries in the Province comply with standards and regulations of the *Air Pollution Control Act.*

21. In January, 1962, Mr. W. B. Drowley, Chief, Air Polution Control Service, was advised by Mr. W. B. Fox, Vineland Horticultural Station, that Mr. J. Casina, Sr. had contacted him regarding crop damage on his farm. Other area farmers were reportedly experiencing similar difficulties and, on May 21st, 1962, Mr. Drowley informed ERCO that "we have had complaints regarding vegetation injury, contamination of cistern water, injury to the lips and tongue, and dust allegedly emanating from your operations." A request was made of ERCO by Mr. Dowley to supply information about the amounts, chemical analyses, and physical characteristics of the materials being emitted to the atmosphere and the steps ERCO was taking to correct and control emissions.

22. On October 18th, 1962, an inspection of the ERCO plant was made by Mr. F. Brisco, Industrial Safety Officer, Ontario Department of Labour, and Mr. H. Nelson of the Industrial Hygiene Branch, Department of Health. At that time working conditions were found to be good. Dusty places were noted around the fertilizer crushing area. The problem of adequate ventilation was reviewed. It was decided at that time that air sampling for fluorides would be carried out by the Industrial Hygiene Laboratory just as soon as the cold weather necessitated that doors and windows in the plant be kept closed. The air sampling, carried out on December 5th, 1962, showed high fluoride levels in the fertilizer plant and especially in the "den" and storage areas of that plant.

23. A follow-up inspection of the plant was made on February 5th, 1963, to check on the previously recommended improvement in plant ventilation and to obtain further air samples for fluoride tests. The plant officials were constantly aware of the necessity of controlling the "fluoride hazard." They modified plant equipment and added "scrubbers" to cut down the emission of fluorides.

24. At a meeting on October 25th, 1963, ERCO discussed its present operations and outlined proposals to recover "dust losses" from rock grinding and granulation operations. Provisions were to be made for the purchase and installation of such new equipment as might be required, as well as for modifications in processing which would further reduce "dust losses."

25. Further liaison was maintained between the Air Pollution Control Service and the ERCO plant, where "emission" information was outlined in detail (by ERCO) and assessed by the Air Pollution Control Service. Mr. Drowley stated that "the largest

single emission was from the phosphoric acid concentrator and that, in our opinion, the emissions were taking place in the form of a mist rather than gaseous [form], hence the lack of distinctive injury pattern except on very sensitive indicators such as wild grape and gladioli."

26. As a result of visits by the Department of Health and ERCO officials to Florida (the site of a complex of phosphate fertilizer plants), ERCO applied for permission to install "scrubbers" on the effluent of the phosphoric acid concentrators. Approval was granted by the Department of Health on December 7th, 1965.

27. On October 22nd, 1965, the Industrial Hygiene Branch of the Department of Health (re-named the Environmental Health Branch in 1967) began a series of studies relevant to the Port Maitland "fluoride problem." These studies were carried out by Dr. E. Mastromatteo, Assistant Director of the Industrial Hygiene Branch, and Dr. V. L. Tidey, Medical Officer, Industrial Hygiene Branch.

28. The demonstrable evidence of damage to livestock and vegetation in the area in 1965 led the Ontario Federation of Agriculture to show an active concern and interest on behalf of local farmers. The Department of Agriculture and Food likewise exhibited its active concern—a concern which was equally shared by ERCO. Arrangements were made that the residents in the so-called "polluted" area would submit claims for damages to an arbitrator agreed to by the Ontario Department of Health and a local committee known as the "Air Pollution Committee for Moulton and Sherbrooke Townships." The claims, as approved by the arbitrator and assessed on the basis of generally accepted terms of reference, were to be paid, without further question, by ERCO. These claims, as was later shown, included loss of cows, heifers, yearlings, calves, sheep, hogs, poultry, compensation for loss or decrease in production of milk, size of litters, infertility, cereal grains, silage crops, forage crops, vegetable crops, shrubs, loss of honey bees, etc.

29. Awards paid by ERCO in 1966, for damage and losses claimed for the crop year 1965, amounted to $86,206.44 and, in 1967, the total paid for the crop year 1966 was $122,126.79. The claims submitted in 1968, for losses in the crop year 1967, although not yet assessed at the time of writing, total $180,709.60.

30. On July 12, 1966, an application was received by the Air Pollution Control Service from ERCO for the installation of ad-

ditional "scrubbing" devices on the granulation plant. Approval was given on July 21st, 1966, for such an installation.

31. In order to assist ERCO in developing as complete a control system as possible, the Air Pollution Control Service contracted with Resources Research Inc. of Lakeland, Florida, to test all the equipment venting fluorides to the atmosphere and report on the total fluoride emissions from the ERCO plant. This consulting firm was selected because of its experience in testing similar installations in Florida. This work was carried out during the period of May 22nd to 30th, 1967, and a report was filed in late June of that year.

32. On July 19th, 1967, Mr. A. Rayner, Chemist, Air Pollution Control Service, who at that time was in the area, reported to the Department of Health, Occupational Health Service, that a resident of the area claimed that he was suffering from fluorosis. This was the first occasion of alleged human fluorosis in the area as reported to the Department of Health. The following day, July 20th, 1967, Dr. V. Tidey visited the area, contacted the individual's physician, and discussed the matter with him.

33. Dr. V. Tidey, on July 20th, 1967, visited the farm of another resident. Lameness, deformed joints, and mouth disease were evident in three cows. The condition was stated as being "fluorosis." The report of that visit also indicated that "an adjacent field of clover was badly burned. Several trees, shrubs and areas of the garden were also burned."

34. On July 24th, 1967, ERCO officials met with officials of the Air Pollution Control Service to discuss the report of Resources Research Inc. Several recommendations for action developed as a result of the report. ERCO was advised that total plant emissions should be reduced to 200 lbs. of fluoride per day and ERCO was asked how they proposed to accomplish such a reduction.

35. Evidence submitted by ERCO showed a total expenditure of over $965,000 for air pollution control equipment in the years 1963 through 1967. But, despite this considerable expenditure (a capital expenditure not increasing production or reducing costs), the Air Pollution Control Service reported, on July 26th, 1967, that the total emission from ERCO was exceeding 200 lbs. of fluoride per day (based on 0.4 pounds of fluoride per ton of phosphoric acid manufactured).

36. It should be noted here that the State of Florida has enforced regulations based on the amount of phosphoric acid pro-

duced by comparable plants in a day, allowing for a percentage of fluoride emissions (in pounds) that is within controllable limits.

This maximum limit is based on four items:

(a) The quantity of fluoride in existence at the time the company decided to expand or modify existing facilities.

(b) The location of the plant.

(c) The latest technology and control devices.

(d) The results from other companies making similar products.

37. On July 26th, 1967, Mr. Drowley stated that achievement by ERCO of a figure of 200 lbs. per day would be no guarantee that "no injury will occur in the area, due to many variables involved in vegetation injury." This statement indicates that "allowable limits" are a compromise between technological capabilities, economic feasibility, and the conditions prevailing in any given area (wind, humidity, temperature, etc.).

38. During the period 1957 to 1967, the Industrial Wastes Division of the Ontario Water Resources Commission (OWRC) was dealing with ERCO on the problem of industrial water pollution in relation to the effluent or water-borne wastes from the plant. This involvement was increased after 1961.

39. The "Air Pollution Committee" mentioned in paragraph 28 and composed of many residents of the "polluted area," sought and obtained a meeting with the Minister of Health. This meeting was held on August 22nd, 1967. The Minister assured the Committee that his Department had been actively involved in the air pollution problem in the area since 1962 and would continue to take any steps necessary to eliminate any hazards in the area. It was his assurance that led to Cabinet approval, on August 24th, 1967, of the establishment by *Order-in-Council* of this Committee of Inquiry.

40. The Honourable, the Prime Minister of Ontario, had already announced that a three-day Conference on "Pollution" would be held on the 4th, 5th, and 6th of December, 1967. At that time some of the great experts in the field of pollution would assemble, discuss the many intricate phases of this important problem, and hopefully shed more light on the complexities of a growing problem —a problem compounded by emotions and economics, facts and foibles, and jurisdictional responsibilities. The deliberations of this Conference would be of inestimable value in the pursuit of the intensified studies contemplated by the Minister of Health. The

Commissioners awaited with interest the results of this Conference.

41. As mentioned previously, the Committee was formally established by *Order-in-Council* on November 6th, 1967, and almost immediately commenced its background studies in preparation for the public hearings and the subsequent preparation of its report.

C. ORGANIZATION AND PROCEDURE

(a) Appointment of Consultants and Commissioning of Studies

42. The literature on "pollution" and "pollutants" to be found in medical, veterinary, agricultural, industrial, biological and other scientific publications is now most extensive; the literature on fluoride alone is very great. A few examples of the extensiveness of such literature are cited:

(a) The Kettering Laboratory, University of Cincinnati Medical School, has compiled abstracts (brief summaries) of the literature on fluorides, running into four volumes.

(b) Publication No. 824 of the National Academy of Sciences —*A Report of the Committee on Animal Nutrition of the Agricultural Board*—lists 135 references when dealing solely with the fluorosis problem in livestock production.

(c) Largent's report on *Fluoride Concentration in Blood and Effects on Enzymes* quotes 204 relevant publications.

(d) Greenwood's *Fluoride Intoxication* cites more than 250 references.

(e) Hodge and Smith in their book *Fluorine Chemistry*, 1965, deal exhaustively with two or three major facets of "fluoride" and list 85 pages of references representing some 3,400 publications.

43. Obviously, then, this report will not contain a complete survey of the literature; it is not the responsibility of the Commissioners to do so. Since there is, in general, major agreement on the results of experiments, surveys and special studies, certainly amongst the recognized and accepted scientists, the conclusions reached by such eminent workers have been taken as the basis for comparison of the evidence elicited at the Committee hearings, where comparisons were relevant.

44. To study in detail the extensive literature in the many and interrelated fields, the Committee commissioned several persons known for their interest and reputation in special areas, as well

as others recognized as experts in a general discipline but without special knowledge of fluorosis, to prepare "briefs" of a summary nature for presentation at the hearings.

45. The material in these briefs, and in other special summaries commissioned by the Committee, is used where deemed advisable, without further reference being made to the source of the material. This practice has been followed wherever extensive use of a brief has been made. Paraphrasing and rewriting would simply delay the presentation of the Report and might well detract from the writing of the Consultants. Using multiple and constant quotations would simply detract from the hoped-for readability of the Report. Each consultant has been advised of our intention. Permission has, of course, been granted to each consultant to use his brief for subsequent publication.

The following are those so commissioned:

(a) Dr. P. J. Lawther, M.B., F.R.C.P., Director of the Air Pollution Laboratories of the Medical Research Council (U.K.), St. Bartholomew's Hospital Medical College, London, England.

(b) Dr. A. E. Martin, M.D., D.P.H., Senior Medical Officer, Ministry of Health (U.K.), Sir Alexander Fleming House, Elephant and Castle, London, England.

(c) Dr. J. L. Sullivan, M.Sc., Ph.D., Consultant, Atmospheric Pollution, National Department of Health and Welfare, Ottawa, Ontario. Consultant on Air Pollution and Industrial Processes.

(d) Professor K. J. R. Wightman, M.D., F.R.C.P., Physician-in-Chief, Toronto General Hospital, and Professor of Medicine, University of Toronto. Consultant in Medicine.

(e) Professor Russell A. Willoughby, D.V.M., Ph.D., Professor, Medicine Section, Department of Clinical Studies, University of Guelph. Consultant in Veterinary Medicine.

(f) Professor S. J. Slinger, B.S.A., Ph.D., Chairman and Professor, Department of Nutrition, University of Guelph. Consultant in Nutrition and Biochemistry.

(g) Professor L. R. Webber, B.Sc., M.Sc., Associate Professor, Soil Science, University of Guelph. Consultant in Soil Chemistry and Soil Pollution.

(h) Professor J. W. Tanner, B.Sc., Ph.D., Associate Professor, Crop Science Department, University of Guelph. Consultant in Crop Physiology.

(i) Professor J. H. Clark, B.Sc., M.Sc., Associate Professor, Agricultural Economics Department, University of Guelph. Consultant in Farm Management and Agricultural Economics.

(j) Professor D. M. Brown, M.S.A., Ph.D., Associate Professor, Department of Soil Science, University of Guelph. Consultant in Crop and Weather Relationships and Agricultural Climatology.

46. Additional consulting experts, under special arrangements, appeared before the Commissioners and added much to the knowledge of the problem. These persons were:

(a) Mr. K. K. Huffstutler, B.Sc., M.Sc., Public Health Engineer in charge, Regional Environmental Facility, Florida State Board of Health; by special arrangement with Dr. Wilson T. Sowder, State Health Officer, Florida State Board of Health. The large and concentrated fertilizer industry in Florida and its problems were considered pertinent to our study. Mr. Huffstutler appeared under the auspices of the Committee but at the specific request of Mr. Brooks (Counsel for the local farmers).

(b) Professor J. W. Suttie, M.Sc., Ph.D., Associate Professor of Biochemistry, University of Wisconsin, Madison, Wisconsin, and collaborator in the extensive studies of the effects of fluoride in the broad field of agriculture. Professor Suttie was retained by ERCO as a consultant.

(c) Dr. V. L. Tidey, M.D., D.P.H., Medical Officer, Occupational Health Service, Ontario Department of Health.

(d) Dr. E. Mastromatteo, M.D., D.P.H., Industrial Health Specialist; Chief, Occupational Health Service, Ontario Department of Health; Certification by the American Board of Preventive Medicine in the field of Occupational Health.

(e) Mr. D. P. Caplice, P.Eng., B.Sc., M.Sc., Director, Industrial Wastes Division, Ontario Water Resources Commission.

(f) Dr. F. C. Nelson, D.V.M., Head, Veterinary Services Laboratory, Guelph Regional Laboratory, Ontario Department of Agriculture and Food.

(g) Mr. D. Bogaerts, Public Health Inspector; Senior Pesticide Control Officer, Pesticides Control Service, Ontario Department of Health.

(h) Mr. W. B. Drowley, B.Sc., P.Eng., Chief, Air Pollution Control Service, Ontario Department of Health; Vice-President, National Air Pollution Control Association.

(b) Procedure of Hearings

47. Counsel for the "Air Pollution Committee," at the request of some members of the Committee, recommended that the Commissioners invite Dr. George L. Waldbott of Detroit, to present evidence at the hearings. An invitation from the Committee of Inquiry, dated January 4th, 1968, was extended to him—18 days before the hearings commenced. Although Dr. Waldbott indicated his desire to appear at the hearings, he did not appear, nor did he submit a brief directly to the Committee. (For more complete details, see Appendix XIV.)

48. The procedure was established that the various consultants would present publicly a general review of the literature relevant to the specific topics under study at the appropriate place in the hearings. On conclusion of such reviews, the consultants, if requested, were to be available for cross-examination, under oath, by Counsel. The Consultants, in addition to preparing a preliminary brief for the Committee prior to the hearings, were to submit a subsequent and final brief, if requested, to the Committee.

49. A fairly comprehensive statement was prepared by the Committee and subsequently used as a Press Release (see the *Preface*). It indicated the scope of the inquiry, the general and specific areas to be considered, and the suggested sequence of events as, at the time, was deemed advisable by the Commissioners.

50. Following the insertion of the required public notices in appropriate daily newspapers (Appendix I), notification of hearing dates having been released (Appendix II), arrangements were made to hold public hearings in the Courthouse situated in the Town of Cayuga, the County seat of the County of Haldimand. Hearings commenced on Monday, January 22nd, 1968, and were concluded on March 13th, 1968. A total of 18 half-days of public sittings were held in Cayuga, where 45 witnesses and consultants were heard. Two half-day public hearings were held in Toronto on March 20th and 21st, 1968, where 10 witnesses and consultants were heard. The official Court Reporters were Nethercut & Young Limited, 48 York Street, Toronto.

51. Members of the Committee, with Counsel and Secretary, and Mr. Deamude and Mr. McAlonan, representing the local "Air Pollution Committee," met with senior personnel of ERCO, together with

their Counsel, Mr. Pepper, and following a presentation of the plant processes, toured the plant. Subsequently the party, excluding some of the ERCO personnel, visited the plant of the Sherbrooke Metallurgical Company and, after a presentation of their processes, toured the various units of the plant. These "tours" took place on January 25th, 1968, and were subsequently recorded in the proceedings of the hearings.

(c) General

52. Between the time of the appointment of the Commissioners and the commencement of the hearings in Cayuga, there was ample opportunity for the Commissioners to read and study the basic sources of information concerning fluorides and their effects on humans, livestock, vegetation, and soils. We also had the opportunity of reviewing the papers presented by many recognized specialists at the Conference on Pollution held in Toronto on December 4th, 5th, and 6th, 1967, and of commissioning the several scientific review studies which have been used extensively in this report. Some of the other scientific literature referred to in the report has been read in the original, others in abstract form, and yet others only in the context of briefs and reviews.

53. Copies of all briefs and other pertinent material were made available on, or very shortly after, their receipt by the Committee, to Counsel for the "Air Pollution Committee" and Counsel for ERCO, in order that they would have available to them the results of the extensive work of the Committee's consultants. Recognizing, too, the necessity for economy, the Committee was pleased to assist Counsel for the local "Air Pollution Committee" by extending invitations to various persons to appear before the Committee and in subpoenaing others that such Counsel might have the opportunity of cross-examination.

54. Final submissions by Counsels were invited by the Committee and at the same time the Committee, prior to the conclusion of the hearings, stated that it would supply to Counsel copies of Professor Wightman's final report (see Chapter II) on the two patients who were still in the Toronto General Hospital, under his care, and copies of Dr. Waldbott's brief. No brief had been received directly from Dr. Waldbott as of July 28th, 1968, the time of writing of the sixth and final draft of this report.

55. Dr. Wightman's final reports on Mrs. Parke and Mr. Boorsma were received on April 3rd and April 18th. Copies were forwarded to Counsel on April 18th, 1968. Concluding submissions

were received from Mr. Brooks, Counsel, under the title "Submission of Property Owners to the Committee of Inquiry on Pollution in the Townships of Sherbrooke, Moulton, and Dunn" and from Counsel for ERCO, Mr. Pepper and Mr. Lamek, under the title "Submission by Counsel on behalf of Electric Reduction Company of Canada Limited." No brief has been received from the Ontario Federation of Agriculture although the Committee advised Mr. Middleton that it would appreciate having such a brief.

56. With very few exceptions the witnesses appearing before the Committee were obviously endeavouring to be factual, to assist the Committee in its inquiry and to present to the best of their knowledge and belief the situation as they saw it and as they believed it to be. This unfortunately cannot be said of all witnesses. Nonetheless, the Committee is deeply indebted to Mr. Brooks and to the persons whom he represented, to Mr. Pepper and Mr. Lamek, who were forthright in their presentations and cross-examinations, to those many other persons who through their evidence assisted materially at the hearings, and to the several Consultants whose assistance was invaluable.

CHAPTER II

Industrial Considerations

A. FLUORIDE-CONTAINING MINERALS AND ROCK

57. Fluorine does not occur in its elemental gaseous state in nature. It is found, in trace to substantial amounts, in igneous and sedimentary rocks as the fluoride ion. The chief fluoride-containing minerals which are used in major industrial processes are:

> Fluorspar (calcium fluoride—CaF_2), cryolite (sodium-aluminia fluoride—Na_3AlF_6), fluoroapatite—$3(Ca_3(PO_4)_2) \cdot (CaF_2)$, and sedimentary phosphate rock which contains about 3.5 per cent F. Fluorspar (or fluorite) is chiefly used as a flux in the smelting of several metals, e.g. steel, and in the ceramic industry. Fluoride compounds are emitted in the production of brick, enamel, and glass. Cryolite is an integral ingredient in the manufacture of aluminum wherein reactive fluoride compounds are produced during the process. Phosphate rock (3.5 per cent F) is the basic source of phosphate in the production of single and triple superphosphate as used for fertilizers and in animal feed supplements.

58. It can be seen that fluoride compounds constitute a potential hazard in many major industries. There are many other industries and processes where smaller amounts of fluoride compounds are used and hazards, particularly to human health, exist. Such a table has been compiled by Dr. D. A. Greenwood of the Department of Pharmacology, University of Chicago, and is reproduced here in part, simply to indicate the variety of processes and the extent of the possible hazard (see Table I)[1]. [Superscript numerals refer to the Bibliography, Appendix XVI.]

TABLE I

USES OF FLUORINE COMPOUNDS IN TECHNICAL PROCESSES

USES	COMPOUNDS
1. Binder for emery wheels	CaF_2
2. Bleaching of cane for chair seats	HF
3. Bleacher in laundries	Na_2SiF_6
4. Cleansing graphite	HF
5. Coagulating rubber	Na_2SiF_6; $MgSiF_6$
6. Decolorizing of glasses	Na_2SiF_6
7. Disinfection of hides and skins	H_2SiF_6
8. Disinfection of hose and tanks in breweries	H_2SiF_6; NH_4F; HF
9. Electrolytic manufacturing of beryllium	Na_2SiF_6
10. Fixing of tannin on cotton in dye works and printing works	SbF_3
11. Flotation of fluorspar and quartz	CaF_2
12. Flotation of lead-zinc fluorspar ores	CaF_2
13. Flux	Na_2AlF_6; Na_2SiF_6; CaF_2
14. Glass etching	HF; NH_4F
15. Hardening cement	$MgSiF_6$; $ZnSiF_6$
16. Insecticidal powders	NaF
17. Making glue, paste and adhesive	HBF_4
18. Making synthetic stones	Sundry compounds
19. Optical industry	CaF_2
20. Preserving wood (building), timber, telegraph poles	NaF; ZnF_2; $MgSiF_6$
21. Recovery from phosphate plant	H_2SiF_6; $PbSiF_6$
22. Refining mineral oils	SO_2F_2; SF_6
23. Refrigerating industry	CCl_2F_2; $C_2Cl_2F_4$
24. Removing sand from castings	HF
25. Rust removal from steel and iron	HF
26. Silicate analysis and condensing agent in laboratory	HF
27. Silk dyeing	HF
28. Synthetic production of cryolite	CaF_2
29. Treating anthracite for making gas coal	HF
30. Window cleaner	H_2SiF_6
31. Woodstaining	CrF_3

59. The original sources of fluoride in soils are: the biotite and muscovite micas, apatite, fluorapatite, hornblende and tourmaline, all of which may occur in the parent material of soils (Dana, 1914)[2], (Dell, 1959 and 1963)[3], (MacLean and Brydon, 1963)[4]. Fluoride is an impurity in Ontario limestones (Goudge, 1963)[5], and in coal where the content ranges from 85 to 295 p.p.m. of fluorine (Thomas, 1961)[6]. It should be pointed out that fluorine, unlike chlorine, is not concentrated in sea water (1.0 to 1.4 p.p.m., Thompson and Taylor, 1933)[7].

60. Most air-borne materials from industries are added to the soils in the nearby area. Thus, if an industrial plant released 10 tons of elemental fluorine into the air per day and half of this settled within a 10-mile radius of the plant, the average fluoride addition to the surface six inches of soil would be about 10 p.p.m. per year (Hansen et al, 1958)[8]. Most of the rock phosphate that is used to produce agricultural fertilizers for the U.S. market and for the Eastern Canadian market, including the basic supplies for ERCO, comes from Florida. The fluoride content of that rock ranges from 3.3 to 4.0 per cent; fluorapatite contains 3.77 per cent fluorine—$3(Ca_3(PO_4)_2) \cdot (CaF_2)$.

B. INDUSTRIAL PROCESSES OF THE ELECTRIC REDUCTION CO. LTD.

61. ERCO manufactures superphosphates, phosphoric acid, and other phosphate compounds, using, as basic raw materials, fluorapatite or natural phosphate rock from Florida, and sulphuric acid. In all cases the Florida rock, as received in the plant, is ground to a fine powder in preparation for the chemical treatments. The "mills" are enclosed but minor escapes of the dust from buildings could occur. These would not be of significance outside.

62. Chemically, Florida phosphate rock corresponds approximately to $3(Ca_3(PO_4)_2) \cdot (CaF_2)$, or 3 molecules of calcium phosphate to one of calcium fluoride. In the dry form, the rock contains about 3.5 per cent fluoride (F).

63. Superphosphate was originally produced in order to increase the solubility of the phosphate in natural phosphate rock so that it would be more suitable for agricultural purposes. In early superphosphate plants, and for many years, only a so-called single superphosphate, containing an available 20 per cent of phosphate, was produced. The reaction for this process is: rock phosphate + sulphuric acid + water = superphosphate + calcium sulphate

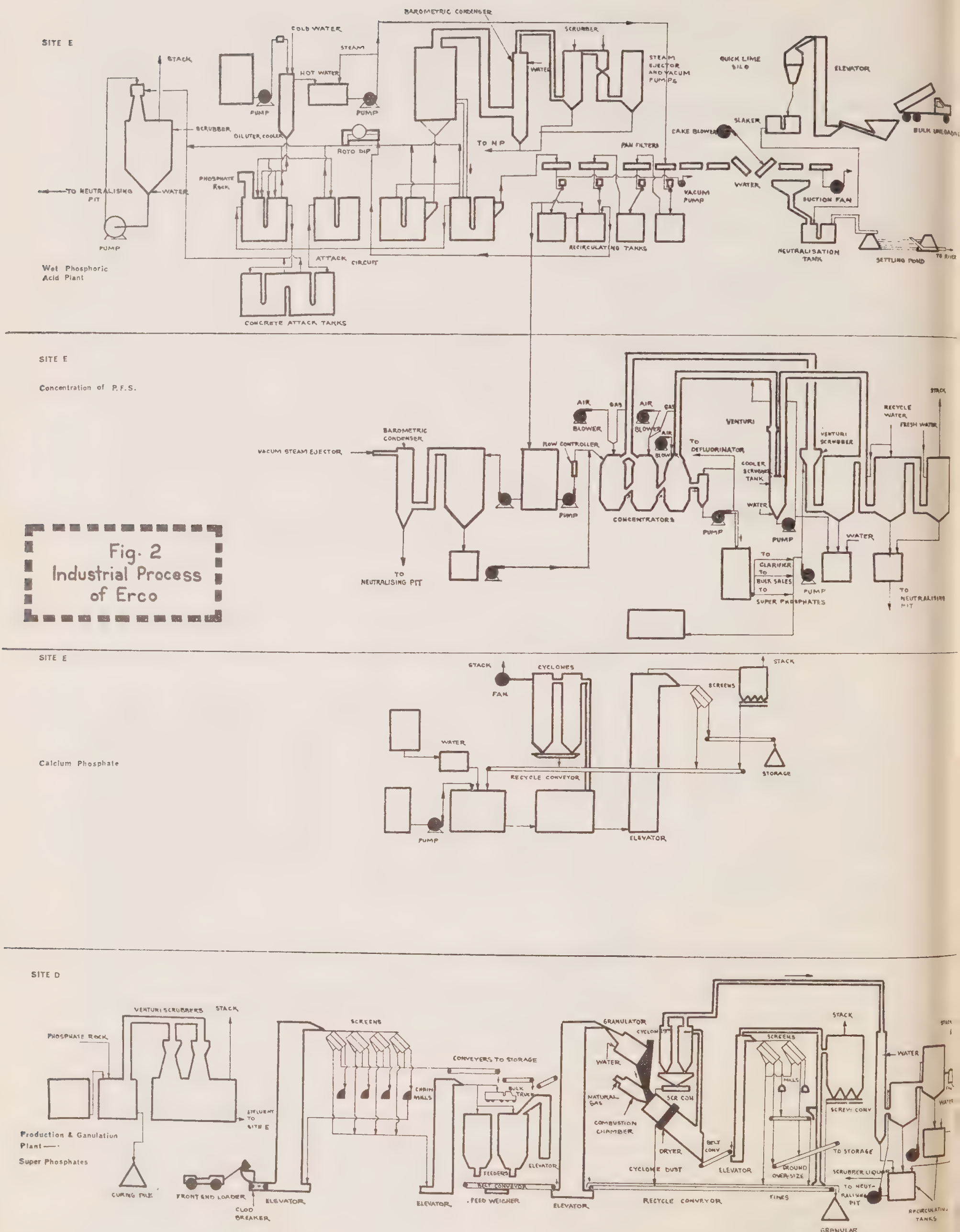


Fig. 2 Industrial Process of Erco

+ hydrogen fluoride: $3(Ca_3(PO_4)_2)\cdot(CaF_2) + 7H_2SO_4 + H_2O = 3CaH_4(PO_4)_2 + 7CaSO_4 + 2HF$ (see Figure 2).

64. Many plants throughout the world, including ERCO at Port Maitland, still manufacture this type of superphosphate. The main part of the industrial operation to cause the above reaction is achieved by mixing the rock phosphate with the sulphuric acid in large wooden box-like containers called "dens." After a sufficient period of time the reaction is substantially complete and the mass in the den has "set," mainly because of the presence of the calcium sulphate or gypsum. The material is then "cut out" and stored in piles for shipment.

65. While the main reaction is going on, part of the calcium fluoride is also decomposed by the sulphuric acid with the resulting production of hydrogen fluoride. This is evolved as a gas but it in turn combines with silica, which is always present in the rock, to produce silicon tetrafluoride and fluosilicic acid. All three of the fluorides as a varying mixture are driven off in volatile form. From old plants, the volatile fluorides were often released to atmosphere, but nowadays it is customary to prevent these emissions as far as possible by "scrubbing" with water. Various designs of scrubber are used, but the most common consists of a tower in which the gases pass upwards against a descending spray of water. In many cases a "packing" technique is used to increase scrubbing efficiency and in more modern plants high energy type systems such as the Venturi scrubber are used.

66. Later developments in the manufacture of superphosphate have tended towards the production of materials containing greater concentrations of available phosphate. One of these, which is made at ERCO, is triple superphosphate containing about 46 per cent phosphate. This product is made by reacting the rock phosphate with phosphoric acid as follows:

$$3(Ca_3(PO_4)_2)\cdot(CaF_2) + 14H_3PO_4 = 10CaH_4(PO_4)_2 + 2HF$$

67. Normally the rock phosphate is mixed with phosphoric acid in a TVA mixing cone and discharged into a den or onto a slow mixing belt to allow for setting. During this stage the treatment of the evolved fluoride gases by scrubbing is essentially the same as for single superphosphate. At ERCO the same "setting" den and scrubbing system is used as for the single product and therefore the two superphosphates cannot be made at the same time.

68. After discharge from the den, the triple superphosphate is conveyed to large curing piles in a special shed where the chemical reaction continues for some weeks. The length of time depends

mainly on the ambient temperature. As the chemical reaction proceeds, more fluorides are evolved. This is one of the disadvantages of triple superphosphate, since it is very difficult to collect and treat the gases given off from the large piles.

69. Phosphoric acid is used for the manufacture of triple superphosphate and is manufactured on the site by reacting rock phosphate with sulphuric acid.

70. As in the manufacture of superphosphate, the fluoride present in the rock also reacts with sulphuric acid and a portion of it is volatilized in the forms described for superphosphate. These could be discharged to the atmosphere but it is usual, as at the ERCO plant, to use scrubbers to absorb as much of the volatilized material as possible.

71. After decomposition of the phosphate rock, which is carried out in special reaction vessels, a mixture of phosphoric acid and calicum sulphate remains. This "slurry" mixture is cooled by vacuum evaporation and then filtered, using horizontal tilting pan filters. This separates the acid from the calcium sulphate (gypsum) which is washed and removed. At ERCO, this material (gypsum) is dumped in a large pond or "lagoon" as waste. It is from this site that particulate fluoride-containing dust may arise.

72. The phosphoric acid is concentrated to the strength required for its further use, as described previously. As made in the reaction vessel, the acid has a concentration of about 30 per cent, which is increased by concentration to about 54 per cent. At the ERCO plant, the concentration occurs in two stages. The first, which is continuous and steam-heated, concentrates the acid to 40 per cent, after which it is transferred to a batch type gas-heated unit. This further concentrates the acid to the desired strength.

73. Before concentration, the acid contains fluoride compounds in solution. These are released with the steam as the acid is heated and becomes more concentrated. This is the major source of volatile fluorides in the manufacture of phosphoric acid and the discharged vapours from the concentrations must be scrubbed efficiently. At the ERCO plant, the scrubbing from the concentration process is achieved through a Venturi scrubber which is followed by a spray tower and a cyclone scrubber. When the phosphoric acid is removed from the concentration process it is simply stored for future use.

74. In addition to the major processes of superphosphate and phosphoric acid manufacture, ERCO also has plants to make cal-

cium phosphate, hydrofluosilicic acid and a plant for granulating superphosphate fertilizers.

75. The calcium phosphate is prepared by reacting locally obtained limestone with phosphoric acid. As the acid still has some fluorides in solution, these must be removed because the final product is used as a food supplement. The removal of the fluorides is accomplished by stripping with steam which is then passed through a scrubber. This reduces the quantity of fluorides discharged to the atmosphere.

76. As the rest of the process only consists of the reaction of the limestone and the stripped acid, no further evolution of fluorides occurs. Some dust is produced as the calcium phosphate is dried. This is removed as much as possible by cyclone collectors and the residual amount passing to atmosphere is not significant.

77. Other manufacturing processes in the plant are not considered to be sources of potential contaminants. Fluosilicic acid is manufactured on a small scale and the possibility of the escape of volatile fluorides is small. In the case of the granulated fertilizer plant, prepared superphosphate is used and no chemical reactions are involved. However, the drying of the superphosphate causes some dust and fluorides to be evolved and the gases from the dryer are treated with cyclones and a scrubber.

C. SULPHUR DIOXIDE AS AN INDUSTRIAL BY-PRODUCT AND POLLUTANT

78. Sulphur dioxide is a relatively stable, non-inflammable, non-explosive, colourless gas. It is highly soluble in water, with which it reacts to form sulphuric acid. Sulphur dioxide is a very common gas compound in urban atmosphere, since this gas is produced whenever elemental sulphur or sulphur-containing compounds are burned in the air. It is perhaps the most widely encountered and best known "irritant gas," not only because of its wide usage but also because of its frequent occurrence as an undesired by-product in the smelting of sulfide ores, in paper manufacture, in the combustion of sulphur-bearing coals and petroleum fuels, and in the action of sulphuric acid on reducing agents. Sulphur dioxide is one of the most prominent gases contributing to atmospheric pollution in large cities and in areas surrounding smelters. The following table indicates the total sulphur dioxide emissions to atmosphere from all sources in the United States in 1963[9].

TABLE II

SULPHUR DIOXIDE EMISSIONS TO ATMOSPHERE: U.S.A.: 1963

	Sulphur Dioxide	
Combustion of Coal	*Tons*	*Per cent of total*
Power Generation (211,189,000 tons)	9,580,000	41.0
Other combustion (112,630,000 tons)	4,449,000	19.0
Sub-total	14,029,000	60.0
Combustion of Petroleum Products		
Residual oil, power generation	650,600	2.8
Residual oil, other combustion	3,052,000	13.1
Other petroleum products	1,115,000	4.8
Sub-total	4,827,000	20.7
Refinery operations	1,583,000	6.8
Smelting of ores	1,735,000	7.4
Coke processing	462,000	2.0
Sulphuric acid manufacture	451,000	1.9
Coal refuse banks	183,000	0.8
Refuse incineration	100,000	0.4
TOTAL	23,370,000	100.0

79. The above table indicates that sulphur dioxide in the atmosphere is primarily a by-product of coal and petroleum combustion (over 80 per cent of total SO_2 emissions).

80. Sulphur dioxide is also used in industrial processes as a constituent in the manufacture of sulphuric acid, sodium sulphite and other chemical compounds. Large quantities are also used in refrigeration, in bleaching, fumigating and preserving. It is used as an anti-oxidant in melting and pouring magnesium where it is applied as a process ingredient. Sulphur dioxide is also used to prevent oxidation in controlled-atmosphere heat ovens in the manu-

facture of magnesium. As an industrial process ingredient, sulphur dioxide is generally an in-plant concern and does not normally constitute an external air pollution problem, since it either reacts with other process ingredients or is dissipated as a treatment gas.

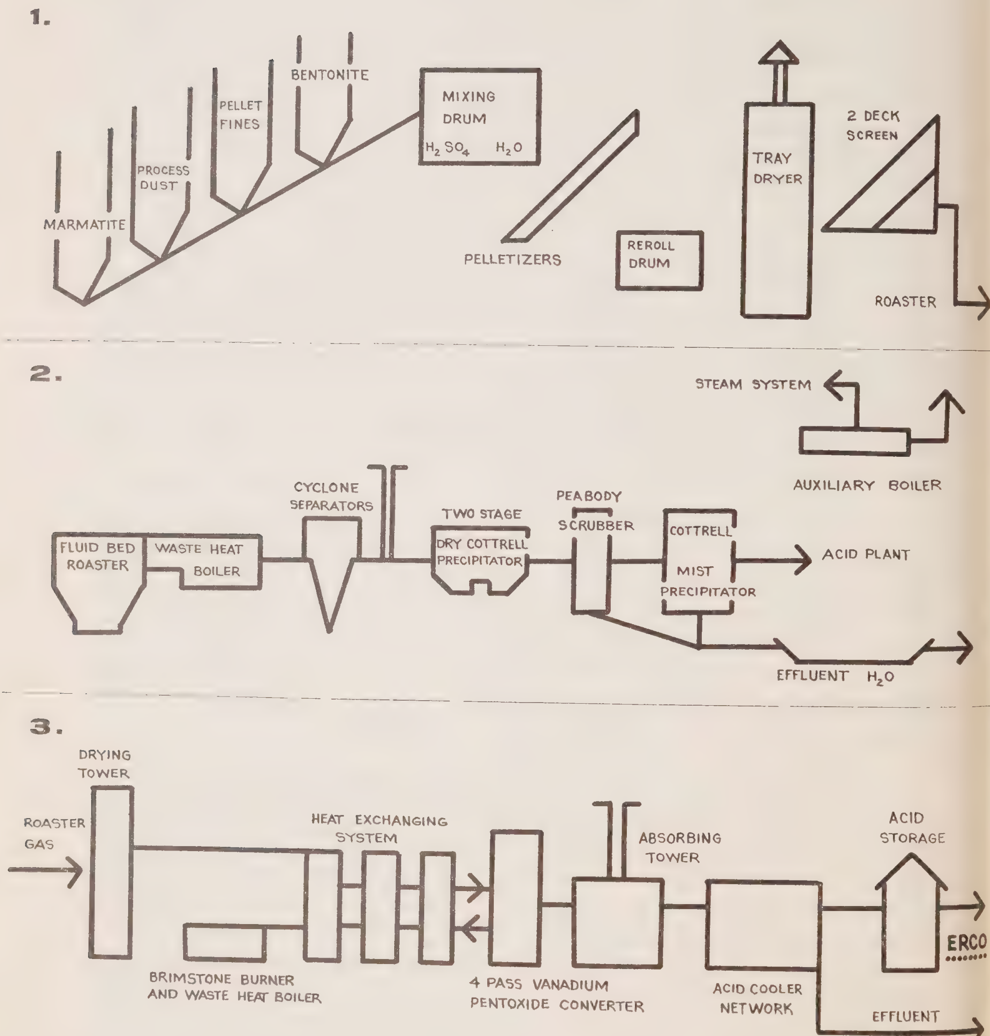
81. Sulphur dioxide, since it is an "irritant gas," can present a problem as a potential health hazard. Exposure to 6 to 12 p.p.m. SO_2 causes immediate irritation to nose and throat and 20 p.p.m. can cause severe eye irritation. Although sulphur dioxide dissolves readily in the human system, its inhalation does affect the upper respiratory tract and bronchii and can cause oedema (pitting) of the lungs or glottis and can produce respiratory paralysis. There is great public concern today with the possible effects of air pollutants on human health and there are those who claim sulphur dioxide is the direct cause of bronchitis, pneumonia, influenza, emphysema, etc. Current medical evidence does not seem to support these claims, but it should be mentioned that significant exposure to pollutants by people suffering from respiratory ailments will cause an immediate acceleration of existing difficulties. It may be stated that exposure to pollutants can contribute to the possible death or decline in health of individuals suffering from chronic respiratory ailments, but to say that the pollutants created the original disorder is not accurate. (The effects of SO_2 on soil and vegetation are dealt with in Chapter IV.)

82. Since the burning of sulphur-containing fuels creates the highest potential SO_2 hazard, some authorities suggest that natural gas and atomic power plants should replace coal and other fossil fuels, thereby reducing the greatest source of SO_2 in the atmosphere. The additional cost of using newer fuel agents creates economic barriers to their substitution in power plants. Most authorities concerned with this problem try to reduce the amounts of SO_2 that are present in any given area at ground level.

83. In order to reduce, as much as possible, the ground effects of SO_2, manufacturers concentrate on installing elevated stacks or chimneys so that the effects of the emissions are reduced because of a much wider dispersal of the pollutant. In most parts of the world today, stack heights are designed according to precise calculations which attempt to maintain maximum ground concentrations below a given acceptable figure. Many air pollution control agencies insist on such design standards before permission is given to build industrial and heating plants.

84. In Ontario, authority to insist upon minimum stack heights and other standards was not possible until the passage of the *Air*

Fig. 3 Industrial Process of Sherbrooke Metallurgical



Pollution Control Act, 1967. In the future, all power plants and industrial operations must meet the Department of Health standards, or they will not be allowed to begin or, as the case may be, continue their operations. Areas such as Hamilton, Sudbury, and Toronto have had serious problems with heavy SO_2 concentrations in atmosphere, and concerted attempts are being made to curtail, as much as possible, the widespread occurrence of this pollutant.

D. INDUSTRIAL PROCESSES OF SHERBROOKE METALLURGICAL COMPANY LTD.

85. The Sherbrooke Metallurgical plant at Port Maitland produces sulphuric acid which the plant supplies to ERCO for the manufacture of agricultural chemicals. During this process, the raw material source of zinc sulphide (marmatite) is converted by roasting to zinc oxide, a metal which is shipped to the parent company (Mathiessen-Hegeler) in West Virginia where it is reduced to metallic zinc (Figure 3).

86. At the Sherbrooke Metallurgical plant, the zinc sulphide ore is initially mixed with water plus processed dust and a binding agent (bentonite) and formed into small pellets. These pellets are dried at a temperature of about 350°F. After drying, the pelletized material is then screened and fed to a fluid bed roaster for oxidation to sulphur dioxide. This roaster operates at a temperature of about 1,832°F. which is maintained by the exothermic reaction of the roasting zinc sulphide.

87. In order to manufacture sulphuric acid, it is necessary to produce sulphur dioxide. This gas is evolved during the roasting of the zinc sulphide according to the reaction:

$$2ZnS + 3O_2 = 2ZnO + 2SO_2$$

88. As it is necessary to supplement the sulphur dioxide obtained from the zinc sulphide, the plant also uses brimstone or native sulphur which is simply burned in a normal roaster:

$$S + O_2 = SO_2$$

89. These two sulphur dioxide streams are then combined for conversion to sulphur trioxide by vanadium pentoxide catalysis:

$$2SO_2 + O_2 = 2SO_3$$

90. Finally, the sulphur trioxide is absorbed in sulphuric acid to which water or more dilute acid is continuously added to maintain a concentration of 98 per cent of the acid:

$$SO_3 + H_2O = H_2SO_4$$

91. The process as described above is a relatively standardized procedure used throughout the world. Sulphur dioxide is produced

in order to be converted into sulphur trioxide, and this liberated SO_2 passes through various cleaning stages consisting of a centrifugal dry collector (cyclone), a dry electrostatic precipitator, a tower scrubber (Peabody) and a wet electrostatic precipitator. In spite of these precautions not all of the sulphur dioxide is converted to sulphur trioxide. The reported efficiency of conversion for the Sherbrooke Metallurgical Plant is 97-98 per cent, a normal figure for this type of process.

92. Under normal operating circumstances, the only gaseous discharges, apart from the boiler system, come from the pelletized ore dryer and the final outlet from the absorber to the sulphuric acid plant. However, when the plant is shut down for an overhaul or due to a mechanical breakdown, provision is made for the immediate discharge of accumulated gases. During these conditions, it is possible for very large amounts of SO_2 to be emitted to atmosphere for a brief period of time.

93. The three main points where some air emissions and thus potential sources of air pollution could be expected are from the pelletizing dryer, the fluid roaster when the relief stack is opened, and from the sulphur trioxide absorber. In each case, the main contaminant which need be considered is sulphur dioxide, although a certain quantity of dust would be emitted continuously from the dryer and intermittently from the roaster. In addition, quantities of zinc sulphite emitted from the plant have been detected in the surrounding area (Appendix III).

94. Sulphur dioxide emissions from the dryer would be very small, probably only amounting to a few parts per million, because the temperature of 350°F. would not be sufficient to cause significant decomposition of the zinc sulphide. As a source of air pollution, this part of the process could be disregarded in this case.

95. From the roaster, the quantity of sulphur dioxide emitted at any time would depend upon the emergency circumstances existing at the time the relief stack is opened. It is, therefore, not possible to predict with any confidence the likely air pollution effects.

96. Certainly, if the relief stack had to be opened while the furnace had the normal charge of ore in it, a massive sulphur dioxide escape would occur since the unreacted part of the charge would contain 30 per cent sulphur. Because of the low height of the relief stack (about 50 feet) the most severe effects would be experienced close to the plant.

97. The effect of sulphur dioxide from the absorber on the sulphuric acid plant can be predicted more accurately because the

escape of the gas is continuous while the process is in operation. Normally, no method of additional absorption is applied to these plants and prevention of adverse effects at ground level is based upon the use of high stacks.

98. At the Sherbrooke Metallurgical works, the daily production of sulphuric acid at normal full production is 700 tons. Assuming an average conversion efficiency of 97.5 per cent (stated to be 97-98 per cent by the Company), the daily emission of sulphur dioxide from the stack would be about 11.4 tons. On the basis of normally accepted diffusion equations, this would produce a maximum ground level concentration of approximately one part per million.

99. In the United Kingdom, the official stack height requirements for a plant manufacturing 600 tons per day of sulphuric acid at a conversion efficiency of 98 per cent is 240 feet. The main stack of the Sherbrooke Metallurgical plant is 120 feet, which would indicate that the ground level concentration is likely to be 4 times higher than permitted in the United Kingdom[10].

CHAPTER III

Human Health Factors

A. EFFECTS OF POLLUTANTS ON HUMAN HEALTH

(a) General Considerations

100. That the members of the Committee, the respective Counsel, and the interested parties in the Community might have a clear, independent, knowledgeable overview of the complexities of the air pollution problem, Dr. P. J. Lawther, Director of the Air Pollution Unit of the Medical Research Council (U.K.), St. Bartholomew's Hospital Medical College, London, England, and Consultant to the Committee, reviewed the problem in a vivid fashion. His presentation as it appears in the evidence is used, in part, directly without further acknowledgement to him.

101. The subject, Dr. Lawther said, is one of extreme complexity, both from the chemical point of view, and the industrial point of view, and certainly from the point of view of its effects on man. His discussion was based on the extensive work which he and his colleagues personally carried out and, as well, on the knowledge which he had gained of the problems in the several countries which he had visited as a World Health Organization Consultant. He was not prepared "to cut it down to the very great simplicities, because it is not a simple subject" . . . and "if some of the stuff that I will show . . . is a little bit complicated, it is only because it is a little bit complicated."

102. The main concern is what the individual is breathing and not what is emitted from power station or industrial stacks. It is

important to differentiate between emissions and ground level concentrations. From a medical research point of view, the most important aspect of the medical effects of air pollution are the particles which can be inhaled, find their way to the lungs and thereby do damage. There is little, if any, concern, from a purely medical research aspect, with particles in emissions which are large enough to fall out and settle on the ground or on vegetation, since such particles are too large to be inhaled into the human lungs. The most dangerous source of air pollution, as far as Dr. Lawther is concerned, certainly in large cities, is the low source which emits chiefly smoke and sulphur dioxide, as a result of the inadequate and uneconomic combustion of fuel oil, coal or gas, or nitrogen dioxide and carbon monoxide in diesel and gasoline exhaust fumes.

103. The sampling of air, the determination of particle size and subsequently of identification, requires the experienced use of many complicated pieces of equipment, from simple and complex samplers to microscopes, thermal precipitators, and electron-microscopes. Since it may generally be assumed that only particles which are less than about 7 microns in size (one micron is one one-thousandth of a millimetre) are capable of getting to the ultimate depths of lungs, the electron-microscope is an essential tool in such studies.

104. Tobacco smoke, like so many other smokes, is a collection of very, very tiny particles of tar—most of them less than 1 micron in size—which, of course, can get right down to the depths of the lungs and are the most powerful irritants. The lung is functional, primarily, at the alveolae level where oxygen and carbon dioxide are exchanged between the blood and the inspired air. As Lawther said, "the surface area available for gaseous exchange in the lung is 90 square meters—the size of a tennis court—so it is a pretty well designed piece of machinery." Dr. Lawther outlined the several different experiments which were being undertaken, in his laboratories and elsewhere, on sulphur dioxide, cigarette smoke, carbon monoxide, diesel fumes, smoke, "smog," etc. He concluded: "This has been a small effort on my part to try and give a sense of perspective to this, to show that it is not any easy problem. It is not necessarily one for the amateur to leap into. There are no simple solutions and this talk is just to show that at last, after many years, we are making headway."

B. EFFECTS OF FLUORIDES ON HUMAN HEALTH

(a) Clinical Considerations

105. Industrial health authorities have, for years, exercised constant vigilance in the identification of cases of human industrial fluorosis. With one major exception, incidents have been mostly of an insignificant character. The exception was in the Danish cryolite industry, where cases were identified and extensively investigated by Røholm in 1937[11]. His descriptions still provide the classic account of severe human fluorosis due to the inhalation of fluoride. He reported that many workers had been exposed to very heavy concentrations of fluoride-containing cryolite dust for long periods of time (30-40 mg. per cubic meter were typical concentrations; in two enclosed areas, concentrations of up to more than 9,000 mg. per cubic meter were found). These enormous concentrations of the fluoride-containing dust produced symptoms of acute fluorosis, the most typical ones being complaints relative to the skeletal and muscular systems, and 35 per cent of the workers complained of pains, stiffness, or rheumatic attacks. On radiological examination, 84 per cent of the exposed workers were found to exhibit evidence of osteosclerosis, the severity being related both to the duration of exposure and the concentration of dust inhaled. Faint or moderate pulmonary fibrosis was found in half of the workers. Røholm also traced, for medical examination purposes, previous workers in the industry. Although they were known to have been exposed to concentrations of dust at least as heavy, or heavier, than were the workers currently employed in the industry, the number and severity of the cases of osteosclerosis were proportionately much less. This was considered by Røholm to be a clear indication of the reversibility of the condition, an observation more recently confirmed by Likins, McClure and Steere (1956)[12], who observed, at Bartlett, Texas, that, when a population ceased to be exposed to a high fluoride intake, fluoride excretion rates continued at a high level for a considerable time.

106. In contrast to the Danish cryolite experience, as recorded by Røholm in 1937, where extremely high levels of fluorides were present, the experience in Great Britain, where they have been dealing with "the fluoride problem" for many years, indicates that, apart from one incident, only occasional cases of human industrial fluorosis have been described and most of those have been without symptoms. Bridge, in 1937[13], described radiological changes in workers at a factory using hydrogen fluoride. Wilkie (1940)[14] similarly found radiological osteosclerosis in two workers employed

in the manufacture of hydrogen and aluminum fluoride and Bowler (1947)[15] reported a similar finding in an employee in a magnesium factory. Murray and Wilson (1946)[16] described, as Dr. Martin called it, an episode where a family, living adjacent to iron-stone workings where calcining took place, was exposed to heavy fumes over a period of years. Members of this family were found to be excreting from 1.6 to 4.6 parts per million fluorine in the urine (acceptable non-hazardous levels 4-8 p.p.m.). They complained of muscular and joint pains, which they were convinced were due to the fumes, but radiological examination revealed no evidence of osteosclerosis.

107. A thorough investigation was carried out, by the Medical Research Council (U.K.), which is referred to as "The Fort William Study."[17] This study dealt with exposure to fluorides of workers in an aluminum factory, and studies which were carried out on them and on persons in the area near the plant. Concentrations of fluorides in the plant atmosphere ranged from 0.14 to 3.43 mg. per cubic meter, in the furnace room, while outside the plant they varied from 0.22 mg., 200 yards away, to 0.04 mg. in the centre of the town of Fort William, Scotland, a distance of 1 mile from the plant. It is interesting to note that none of the factory workers had complained of any symptoms, and the incidence of aches and pains in the furnace room workers was no greater than in other workers in other parts of the plant, or in the local residents. There was a suggestion that the furnace room workers had rather more frequent digestive disorders and coughs and the investigators themselves noted the irritating nature of the furnace room fumes.

108. No dyspnoea on exertion (difficulty in breathing) was found in the workers and no increased liability to suffer from fractures was noted. There were no physical signs of skeletal fluorosis, but one furnace room worker was suffering from ankylosing spondylitis which was thought by the investigators to be unconnected with fluorides. Among the other factory workers, there was one case of chronic pulmonary fibrosis and one of emphysema. Radiological examination revealed signs of osteosclerosis (although no symptoms) in a number of the furnace room workers, the proportion of cases increasing with the time of exposure. Radiological signs included lipping of the dorsal and lumbar spines with beak-like exostoses, a granular amorphous and somewhat dense appearance of the pelvis, often with short bony exostoses, and plaques of dense bone on the tibia and fibula.

109. Workers in the factory showed an increased urinary excretion of fluoride, the amount being closely related to the severity

of exposure, the average excretion of 65 heavily exposed workers in the furnace room being 9.03 mg. per day. No clinical signs or symptoms were found among the local residents of the area. The incidence of mottled teeth in children in the vicinity did not differ appreciably from that in unaffected areas (from Martin).

110. Likewise, very few cases of human industrial fluorosis have been found in other countries—even in those areas where emissions of fluorides exceed what may be described as "acceptable levels." Derryberry and his colleagues (1963)[18] described an investigation in a phosphate fertilizer plant in the U.S. in which a group of 74 workers, exposed to relatively high fluoride concentrations, was compared with a matched group of unexposed workers. The fluoride exposure of the individual workers was estimated by repeated examination of urine samples taken at the end of the night shift, and the percentage of specimens containing or exceeding 4 mg. per litre (approximately 4 p.p.m.) was calculated as an index of exposure for each person. No disability attributable to fluoride was found in any of the workers. Minimal or questionable degrees of increased bone density were found radiologically in 23 per cent of exposed employees, but in no case were the bone changes sufficient for them to have been recognizable as increased bone radiopathy in routine radiological practice. No increase of abnormal findings relating to gastro-intestinal, cardio-vascular, metabolic, or haematological conditions was found in the exposed group. Respiratory conditions, however, were more frequent, though these might have been due to the irritating properties of the acid gases—it is not known whether masks or respirators were worn (from Martin).

111. In the preceding four paragraphs, we have dealt chiefly with the influence of fluorides on in-plant workers who have been exposed for long periods of time to relatively high concentrations at their jobs in the plant. Brief reference should be made to the effects of lesser concentrations in the air in the vicinity of the plant at Fort William, where no appreciable effects were shown. The comparable aspects of our study in the Port Maitland area warrant a further comment on the fluoride intake of people in a so-called affected area.

112. As Martin pointed out in his presentation at the hearings, suggestions have been made that, where water is fluoridated at 1 p.p.m., the additional fluoride intake of the population, in areas where there is industrial pollution, might create a hazard. The same suggestions might be made where the drinking water sup-

plies came from deep wells, where the normally occurring fluoride concentration of the water is, as it is in many localities, higher than the optimal 1 p.p.m. and where there is a potential industrial hazard. Such fears may be discounted, for, in man, the principal hazard, if any, would be in the inhalation of fluorides in the air. The average man engaged in moderately strenuous work is known to inhale approximately 20 cubic meters of air per day. If, therefore, the fluoride concentration of the air of any place is known, it is a simple matter to calculate as an upper limit the maximum fluoride uptake on the simple assumption that all the fluorides inhaled are retained.

113. Martin pointed out that, using this method, it was found that, in Central London (England), the fluoride intake of the average man would be of the order of 0.003 mg. per day and 0.03 mg. during a day of thick fog with exceptionally heavy pollution. Stoke-on-Trent, in the centre of the pottery industry, and Rotherham have been among the most heavily polluted areas of England. In the former, where the pollution was a generalized one covering the entire city and its environs, the intake of fluorides of the average man would be of the order of 0.04 mg. per day (slightly higher than a heavily polluted foggy day in London) and, in Rotherham where the pollution was more localized, the intake would be 0.01 to 0.02 mg. per day. It is evident, therefore, that with these levels of pollution, far exceeding anything in Canada—even when allowance is made for the increased air intake of a workman or a farmer engaged in very strenuous work—the amounts of fluoride absorbed from the atmosphere would be but a fraction of that contained in his ordinary diet (McClure, 1949)[19]. On the other hand, Lindberg (1964)[20] reported atmospheric fluoride concentrations of from 0.098 to 0.458 mg. per cubic meter near a superphosphate plant in the USSR. This would suggest maximum possible adult intakes of from 1.9 mg. to 9.7 mg., and high levels of fluoride intake were, in fact, confirmed by the finding of dental fluorosis and a low incidence of dental caries in school children in that area.

114. Although mention has already been made to "mottling of the teeth," dental fluorosis and its significance should be noted at this point. The condition of dental fluorosis or "mottled teeth" was found to be produced by, or associated with, the ingestion of water containing relatively high concentrations of fluoride. Dental fluorosis has been defined by Dean (1954)[21] as an "endemic hypocalcification and hypoplasia of the enamel. The affection is largely limited to the permanent teeth although in areas of medium to marked

severity deciduous teeth may be mildly affected." Dean, in studies extending from 1933 through 1942, showed that, where the concentration of fluoride in area water supplies was over 4 p.p.m., the degree of dental fluorosis was severe and that teeth showed confluent or discrete pitting. In localities where the natural drinking water contained 2.5 to 3.0 p.p.m. of fluoride, those teeth which were affected had a dull, chalky appearance and later on they developed a characteristic brown stain which increased with age. In areas where the water supply contained 1 p.p.m. of fluoride or less, there was no evidence of "mottled teeth." There is no fluorosis of aesthetic significance associated with a water fluoride concentration of approximately 1.0 to 1.6 p.p.m. Indeed, and this is extremely significant and very well known now, the incidence of dental caries is dramatically lower in a population where the communal water supply contains approximately 1.0 p.p.m. of fluoride.

115. Other possible health hazards of fluorides in concentrations found in industrial emissions are quite remote. As is discussed more fully in a later section of this report, it has been shown that the increase in the fluoride content of milk from cows suffering from readily demonstrable fluorosis is negligible. Likewise, as will also be discussed later, there is no significant accumulation of fluorides in the soft tissues of such animals and therefore no health hazard to the eating of such meat. It has been shown, too, that the prolonged boiling of bones from such animals does not release fixed bone fluorides, so there is no human health hazard in consuming revelant soups and stews (Allcroft, 1956)[22].

116. Although the question of the effects of fluorides on vegetables and other plants will be dealt with in Chapter IV, it is necessary to mention at this time, in connection with human health hazards, that no appreciable increase occurs in the fluoride content of vegetables and fruits grown on soils rich in fluorides and any hazard to health, if it exists, would arise from surface contamination of the vegetables and fruit. On the basis of the known amounts of fluoride deposited in affected areas in the U.K., of the amounts of vegetables consumed by the population in those areas and from the general practice of washing vegetables and discarding the outer and older leaves, it is apparent that any additional intake in the diet would also be negligible.

117. In this latter connection, the following table, from McClure (1939)[23], should be of interest.

TABLE III

FLUORINE CONTENT OF VARIOUS FOODS

Food	*Fluoride p.p.m.*	*Food*	*Fluoride p.p.m.*
(Fluoride reported in food as consumed)			
Milk	0.07—0.22	Pork Chops	1.00
Egg White	0.00—0.60	"Hot Dogs"	1.70
Egg Yolk	0.40—2.00	Round Steak	1.30
Butter	1.50	Oysters	1.50
Cheese	1.60	Herring (smoked)	3.50
Beef	0.02	Canned Shrimp	4.40
Liver	1.50—1.60	Canned Sardines	7.30—12.50
Veal	0.02	Canned Salmon	8.50— 9.00
Mutton	0.02	Fresh Fish	1.60— 7.00
Chicken	1.40	Canned Mackerel	26.89
(Fluoride reported in dry substance of food)			
Rice	1.00	Honey	1.00
Corn	1.00	Cocoa	0.50— 2.00
Corn (canned)	0.20	Milk Chocolate	0.50— 2.00
Oats	1.30	Chocolate (plain)	0.50
Crushed Oats	0.20	Tea (various brands*)	30.00—60.00
Dried Beans	0.20	Cabbage	0.31— 0.50
Whole Buckwheat	1.70	Lettuce	0.60— 0.80
Wheat Bran	1.00	Spinach	1.00
Whole Wheat Flour	1.30	Tomatoes	0.60— 0.90
Biscuit Flour	0.00	Turnips	0.20
Flour	1.10—1.20	Carrots	0.20
White Flour	1.00	Potato (white)	0.20
Ginger Biscuit	2.00	Potato (sweet)	0.20
Rye Bread	5.30	Apples	0.80
Gelatin	0.00	Pineapple (canned)	0.00
Dextrose	0.50	Orange	0.22

*Analysis of tea leaves. A cup of tea contains about 0.12 mg. of fluoride.

118. As will be shown in a later chapter of this report, wherein we deal with the effects of fluorides on plants, it may be stated here in general that, even though fluorides may accumulate in or on the leaves of non-resistant plants, they apparently do not accumulate to any marked degree in the fruit or ripened seed.

(b) General Review

119. Dr. A. E. Martin, Senior Medical Officer, Environmental Health Branch of the Ministry of Health (U.K.), an experienced

worker on the environmental influence of air and water on human health, a consultant to the World Health Organization and a member of many other scientific and research bodies, acted as a consultant to the Committee. In this capacity, he presented, at the hearings, a general review of the effects of fluoride poisoning on human health. In this report, his words have been used directly or have been paraphrased. A few special quotations, however, have been identified.

120. We, all of us, think we know what a poison is: a substance which is harmful to the body, and a substance which may even cause death if we take too much of it. It is not, however, quite as simple as that, because many substances can be essential to life in certain concentrations and yet be poisonous in other concentrations. Oxygen, in the very air that we breathe, is poisonous if one tries to breathe pure oxygen for a time. Even water, if one takes too much, can be poisonous. Common salt, iron, copper—all are substances which are essential to life, but if one gets too much of them, Dr. Martin pointed out, they can be poisonous. The very term "fluorides" suggests a chemical which is perhaps rather fearsome to some laymen, but (as has been pointed out elsewhere in this report) fluorides are very common salts—simple, inorganic salts which occur in the soil, in the atmosphere, in our food and in our drink.

121. In the soil, one usually has several hundred parts of fluorine per million parts of soil, and they may even be much higher than that. In sea water, there is about 0.8 of a part per million and, as a result of that, the tissues of fishes, particularly the bones of fishes, are very rich in fluorides. Fluorides are inevitably present in the human body because they are present, either in trace quantities or in rather large quantities, in most items of diet and in most drinking water.

122. As Dr. Martin pointed out, it is important to remember or to recognize that, when soluble salts are dissolved in water, they dissociate and "ionize." One of the simple salts, sodium fluoride (NaF), is composed of one atom of sodium and one atom of fluorine, joined together. When that molecule of sodium fluoride is dissolved in water, in dilute solution, it will dissociate into two ions, one of sodium and one of fluoride. Similarly, calcium fluoride (CaF_2) consists of one atom of calcium and two atoms of fluorine and, when that is dissolved in weak solution in water, it "ionizes" into one ion of calcium and two ions of fluoride. Thus, the use of the word "fluoride" implies the fluoride "ion" which is the basis

of quantitative and comparative considerations. The term "fluoride" has been used chiefly throughout this report but, since some authors refer to "fluorine," this term has been used in those specific contexts.

123. Incidentally, some writers have, quite unwisely we think, reported results as parts per million of sodium fluoride. Of course, the results are high as compared to the p.p.m. of fluoride and very erroneous comparisons may mistakenly be made. The atomic weight of sodium (Na) is 23; the atomic weight of fluorine (F) is 19: therefore, the molecular weight of sodium fluoride (NaF) is 42. If the fluoride content of a substance is expressed as 10 p.p.m., NaF, this would be the equivalent of 4.7 p.p.m. expressed as fluoride (ion). The fluoride ion, whatever the source, is exactly the same as any other fluoride ion.

124. In considering the effects of fluorides on humans, it is essential to consider the metabolism, physiology, and toxicology of fluorides as they act normally in the human body, and the way they act when there are excessive concentrations of them. It is then necessary to consider health statistics and find out how populations react in the presence of different concentrations of fluoride in their food and drink and in the atmosphere. One should then consider the relationship of fluorides to individual diseases. Dr. Martin did all of these in his presentation.

125. First of all, fluorides may be absorbed from the air, since fluorides are present in ordinary coal smoke and in the air surrounding certain industries. The most usual route of absorption, however, is the alimentary tract, either in food or drink. The fluoride ion, if it is in a soluble compound, is absorbed fairly quickly from the gut into the bloodstream. Inside the body, it is rapidly distributed in the blood and tissue fluids and, within a short period of time, some of it is taken up by the bones and a considerable portion (50 per cent perhaps) is excreted through the kidney into the urine. Thus, urine and bone fluoride concentrations are very relevant to the amounts of fluoride ingested. The body of a person who is consuming a fairly constant level of fluoride, say 1 p.p.m. in water and additional fluorides in the normal diet, approaches a state of relative balance in which the amount of fluoride ingested into the body roughly equals the amount which is being excreted in urine, faeces, perspiration, and hair, tears, etc.

126. If a person, having lived in a high fluoride area and having ingested much fluoride in the drinking water, suddenly moves to a "low fluoride" area, then the bones will give up some of their

fluoride into the bloodstream which will, in turn, be excreted in the urine and, in the process, the concentration in the bones will then begin to decline. In the U.K., and presumably in Canada and the United States, based on experiments in the U.K., there is a gradual increase in bone fluoride. At age 10 there is very little but, as the person grows older, the amount of fluoride in the bone increases; however, as late middle age is reached, the fluoride content tends to level off. This same phenomenon occurred in areas where there was a low fluoride content of the drinking water (0.2 p.p.m.), in an area where the content of fluoride in the water was 0.8 p.p.m., and as well in an area where the water contained 1.9 p.p.m. fluoride.

127. The body has the remarkable ability of retaining a constant level of fluoride in the blood plasma. That level is maintained —whatever the intake of fluoride, up to a certain level—and, if a population is living in an area where there is very little fluoride in the water and where they are not getting much fluoride in their diet, then the blood maintains the same level of fluoride as in an area where there is, say, 1 p.p.m. of fluoride in the water. (The 1 p.p.m. of fluoride in municipally-controlled water supplies is not just a figure arrived at arbitrarily; it has a specific physiological and metabolic significance.) However, if the amount of fluoride intake goes up considerably, then the amount of fluoride in the blood plasma increases. Dr. Martin illustrated this significant point by pointing out that, with amounts of fluoride ingested (in this case in drinking water) between 0.15 and 2.5 p.p.m., the level of fluoride in the blood plasma was maintained at 0.14 to 0.19 p.p.m., but then in an area where there was a greater amount of fluoride in the drinking water, 5.4 p.p.m., the amount of fluoride in the plasma increased to 0.26 p.p.m. At no time is there more than 10 per cent of the amount of fluoride that has been taken by mouth circulating in the blood. It has been eliminated, explained Dr. Martin, from the blood at the same speed as it has been absorbed from the intestines.

128. This, of course, leads to another interesting thing: the fact that the soft tissues of the body have a fluoride content, but the fluoride content is related to the amount of blood and tissue fluid in that particular tissue. Thus, a vascular organ, such as the liver or the thyroid gland, will have a higher level of fluoride in it than there is in a soft tissue which consists of fibrous tissue, e.g., muscle. The fact that the fluorides are absorbed into the bones gives a clue to the picture of what are likely to be the toxic effects

of fluoride if excessive quantities are taken by mouth or by breathing.

129. At this point one must consider the biological effects of fluorides and, in doing so, it is necessary to distinguish first of all between the effects of organic fluorides and those of inorganic fluorides. In the former group of chemical compounds, the biological effects are in most cases not due to, or associated with, the fluoride ion. In the latter group, the inorganic fluorides, the biological effects are almost entirely due to the action of the fluoride ion (F). Secondly, it is necessary to distinguish between the effects of the ingestion of a massive dose or doses of fluorides, which produce acute fluorosis, and the ingestion of much smaller quantities of fluorides over a long period of time, which may produce chronic fluorosis. In this Port Maitland study, we are dealing only with the effects of inorganic fluorides, and these only on a quantitative-time basis, which may result in a demonstrable effect of chronic fluorosis. There are no organic fluorides involved nor is there any possibility of acute fluorosis being a factor.

130. Dealing briefly with the acute effects of inorganic fluorides, it may be stated that "after a large dose of a soluble fluoride is ingested, accidentally or by suicidal attempt, there follows a catastrophic symptom-train. The course of poisoning is violent and brief; deaths are frequently recorded in 2-4 hours." A "large" dose means a teaspoon or more (5-10 grams) of sodium fluoride. The exact cause of death is still obscure, according to Hodge and Smith (1965)[24]. Sixty such fatal cases of acute fluoride poisoning, reported during the period 1873-1935, were summarized by the great scientist and world-renowned fluoride specialist, Kaj Røholm, (1902-1948)[11]. Hodge and Smith summarized the cases of acute, fatal fluoride intoxications, where such cases were reported in sufficient detail, for the period 1935-1957, and in addition reported the number of deaths from sodium fluoride in the United States in relation to the total number of deaths from poisons—in summary, as follows[24]:

TABLE IV

DEATHS FROM SODIUM FLUORIDE IN RELATION TO DEATHS FROM OTHER POISONS

(1933-1935)

Years	*Deaths from Sodium Fluoride*			*Deaths from Solid or Liquid Poison*		
1933-1935	*Accident*	*Suicide*	*Total*	*Accident*	*Suicide*	*Total*
	138	408	546	17,480	39,839	57,319

131. A massive dose of soluble fluoride compounds is required to produce these fatal results or even acute non-fatal intoxications —quantities which are never, or could ever be, remotely associated with fluoride emissions from industrial plants or the most contaminated water or vegetation.

132. In China, India, Japan, and in some instances in Africa and the Middle East, cases of acute fluorosis have been reported, but these were the effects of consuming over a long period of time grossly excessive quantities of fluoride as found in some drinking water sources in the areas, and are in no way relevant to situations in the U.K., the U.S.A., or Canada. Acute fluorosis has been mentioned here only so that it may immediately be dismissed from our minds. It has no relevance to the Port Maitland study.

133. The condition of fluorosis, sometimes known as chronic industrial fluorosis, and also called "Crippling Fluorosis," was first identified and described by Møller and Gudjonsson in 1932[25]. This condition has been described many times since, both in animals and in man. It is a crippling and painful disease in which the affected person "slowly but progressively becomes debilitated until he can no longer carry out simple physical tests. Once fully developed, the disease is not rapidly reversible; in severe cases, the damage is to all intents permanent." (Hodge and Smith, 1965)[24].

134. This condition of "crippling fluorosis" developed after exposures to relatively high concentrations of fluoride over long periods of time. The consensus of medical and other scientific opinion would indicate that, in man, some 20-80 mg. or more of fluoride ingested into the body, through air, food and/or water, daily for periods of years (10-20), will eventually produce chronic or "crippling fluorosis." Throughout the acceptable scientific literature, it is recognized that "the crippling effects are limited to the skeleton." An outstanding sign is the "poker back," a permanent stiffness of the spinal column from extensive calcification of the broad ligaments running down the spinous processes. Joints become painful and disabled. The bones frequently appear hypercalcified, although a mixture of hyper and hypocalcification is sometimes observed, giving the bones a "moth-eaten" appearance that could be confused with certain forms of bone cancer. Exostosis (thickening of the outer surfaces) appears on the long bones and elsewhere (e.g., pelvis). The earliest detectable changes are found by radiographic examination: a hypercalcification or radiopacity characteristically first observed in a "pelvic vertebra." (Hodge and Smith)[24].

135. With great advances in industrial practices and air pollution control, potential "industrial" or "crippling fluorosis" should be detected years before any serious effects are noted. It is common practice—and indeed a precautionary necessity—that urine samples of men working in fluoride-emitting plants be taken from time to time and analyzed for fluoride concentrations. There are accurate quantitative methods of analyzing the fluoride content of urine. The results are usually corrected to a specific gravity of 1.030 and reported as so many parts per million of fluoride, e.g., 1 p.p.m.F.

136. Reference will be made later (paragraph 149) to the observations in the "Fort William Study" where the only positive finding among the furnace room workers (fluoride-containing fumes and dust), as compared to other workers in and outside the plant and persons living and working at a distance from the plant, was a mild but still typical picture of some excessive deposition of bone as indicated by x-ray (see also paragraph 108)[17].

137. For further evidence of the effects of ingestion of relatively high levels of fluoride (and one must remember that fluoride is fluoride regardless of the source), Dr. Martin made reference to a classic study in the United States, where a survey was made between the two towns of Bartlett and Cameron (see also Morden report 1960)[26]. The town of Bartlett had 8 p.p.m. of fluoride in its municipal water supply and the town of Cameron had only 0.4 p.p.m. The study covered a period of 10 years. Medical histories, physical examinations, x-ray examinations, dental examinations and laboratory examinations were conducted on a group of people from each town at the beginning of the period and again at the end of the 10-year period. As expected, an increased amount of mottling of the teeth was found in the people of Bartlett (ingesting high fluoride levels) but there were no significant differences between the two areas in any of the complaints, any symptoms, or in physical examinations. As in the "Fort William Study," excessive deposition of bone was found in some of the Bartlett people, but it was not causing any physical signs or physical disability to the people concerned. It should be recalled that the Bartlett people were consuming water with a fluoride level of 8 p.p.m. for a minimum of 10 years! Translating this concentration of fluoride into total intake, those people had been ingesting, in the water alone, about 16 milligrams of fluoride per day, and much more on hot days (Bartlett is in Texas) when more water would likely be consumed.

138. Dr. Martin pointed out that a similar investigation had been carried out in Russia between two towns, one with 4 p.p.m. fluoride in its water supply, and one with a low water fluoride concentration. No difference could be found in the incidence of illness or in the complaints or in the physical examination of the people. Dental mottling, of course, was present in the people in the "high fluoride water" town.

139. It is interesting, too, that in another investigation carried out in Texas and Oklahoma—where there are at least 320 towns with high fluoride water—170,000 x-ray plates were examined. Only 23 cases of osteosclerosis were found. Each of those was subsequently given a complete physical examination, their medical histories carefully taken and again no evidence could be found of any physical disability, even though all of the people were consuming water containing between 4 and 8 p.p.m. fluoride.

140. All of the above studies show that, in humans, bone changes occur first and that there are no detectable, physical disabilities or symptoms experienced by persons ingesting even relatively high levels of fluoride. Other investigations in the U.S.A., covering 32 pairs of towns, where a "high fluoride town" was paired with a "low fluoride town," conclusively showed that no differences could be found in the total death rates from all causes or in the death rate from heart disease, cancer, and nephritis.

141. Reference was also made by Dr. Martin to a further study in the U.K., where it was shown that a high atmospheric fluoride level existed around Rotherham and in Stoke-on-Trent, these being two of the most "polluted" areas in England at that time (1960-1961). At Rotherham, the amount of fluoride in the air was 0.7 micrograms per cubic meter. This amount was sufficient to cause extensive fluorosis among cattle, but the calculations showed that the maximum absorption by a man living and working in the area throughout the 24 hours was 0.01 to 0.02 of a microgram—much less than the amounts of fluoride in a single cup of tea. In Stoke-on-Trent, the amount was 0.04 of a milligram—again, much less fluoride than in a single cup of tea!

142. Expressing this type of situation in another way, it has been found in the U.K. that fluoride deposited in rural areas in England (e.g., in Essex) averages 0.69 grams (g.) of soluble fluoride per 100 square meters per month, whereas in London values ranging from 0.58 to 2.6 have been obtained, the excess over rural areas presumably being largely due to the combustion of coal. Measurements in Warwickshire have varied from 0.5 to 1.08 g.

per 100 square meters, the latter being recorded downwind from an aluminum plant. In contrast to these readings, a monthly average of 4.5 was measured near the centre of Rotherham, approximately 1½ miles from a steel mill which was at the time using fluorspar in open hearth furnaces, and an average of 3.64 was obtained at a site near the centre of the pottery industry in Stoke-on-Trent (Martin, 1968)[27]. Similar, and in some cases somewhat higher, atmospheric concentrations of fluorides have been reported from urban and rural areas of the United States (Cholak, 1960)[28].

143. The relative unimportance of atmospheric fluorides as a human hazard has also been demonstrated in a recent study by Call and his colleagues (1965)[29] of material from 127 autopsies in the state of Utah where air pollution from industry was known to give rise to atmospheric fluoride concentrations of up to 0.8 micrograms (μg.) per cubic meter of dry air, with a mean annual value of 0.24 μg. (from Martin). These levels of fluoride did not produce any gross or even histological changes in the bones or soft tissues.

144. The atmosphere of London, England, contains fluorides due to the burning of coal. Measurements, carried out by Dr. Lawther's laboratory, showed that the amount was 0.1 to 0.15 μg. per cubic meter of air, which in turn would give an intake of 0.003 of a milligram of fluoride per day from the air—indeed "a very tiny amount." Even in foggy weather in London, the amount went up only to 0.3 of a microgram per cubic meter of air. In one recorded case in Russia, where a factory, apparently without a process system for scrubbing the fumes, was emitting very excessive amounts of fluoride fumes, the pollution in the vicinity of the factory was very considerable. Dental mottling was found in the children, and the amount of fluoride absorbed was from 1.9 to 9.7 milligrams per day.

145. From an extensive review of the literature, and from Dr. Martin's own personal experience and involvement in relevant studies, he concludes: "summarizing, we have the position that gross excess of fluoride intake such as occurs in some areas in the Far East will cause very serious disease. In investigations which have been carried out in Denmark, Britain, the United States and in Russia, we are able to indicate to a certain degree of accuracy that there are levels which will cause signs radiologically (by x-ray examination) but which do not cause physical disabilities and do not cause symptoms among the population."

146. Let us turn again, for a moment, to industrial emissions. It is well known, of course, that traces of fluorides derived mainly

from the combustion of coal and other fuels are to be found in all urban atmospheres. Localized areas of heavier pollution occur in various parts of the world as a result of certain specific industrial processes and may have a serious effect on agriculture. The extent of such emissions from industrial processes was emphasized by Dr. A. E. Martin when he said that the major sources of fluorine compounds in the atmosphere are well known and that, in 1961, it was estimated that some 25,000 tons expressed as fluorine were emitted annually in England and Wales. Twelve thousand tons were derived from the industrial use of fluorspar (of which 10,000 tons were emitted during the manufacture of steel), 5,000 tons from industrial and domestic use of coal, 4,500 from the heavy clay industry, 600 tons from the pottery industry. "Emissions from blast furnaces, from the chemical industry and from the manufacture of hydrofluoric acid, fertilizer, phosphorus and zinc production were negligible."

147. Since the special concern of this Committee is the effects of "pollution" in a specific area in which are situated two industrial plants, one producing zinc and sulphuric acid and the other producing hydrofluoric acid, phosphoric acid and phosphate fertilizers, it is well to emphasize Dr. Martin's very relevant and comparative statement—"Emissions from . . . the manufacture of hydrofluoric acid, fertilizers, phosphorus and zinc production were negligible." However, the figures cited in paragraph 146 are of limited value, since it is the localized area of high pollution which may constitute the hazard. Moreover, as Dr. Martin pointed out, the fumes may be discharged from tall chimneys giving satisfactory dispersal or, alternatively, they may be discharged at a low level with resulting heavy ground pollution. Heavy particulate matter will be deposited in the immediate vicinity of the emissions, whereas aerosol particles and gases will be dispersed over a wide area and a proportion will diffuse upwards and not reach ground level.

148. It is apparent, therefore, that hazards may exist either from the deposition of fluoride particles on the ground and on herbage or from the presence of fluorides in the atmosphere, where they may be inhaled by animals or man. Crops may be damaged and plant growth restricted by either, but it is the physical contamination of herbage which gives rise to the principal hazard, animal fluorosis.

149. Although it becomes obvious that many studies and long-term experiments have been carried out by recognized investiga-

tors, specific reference has already been made to one study which is considered to be especially relevant. This is a well-documented report (Medical Research Council, Memorandum No. 22, *Industrial Fluorosis*)—a study of the "Hazard to Man and Animals" near Fort William, Scotland, and published by His Majesty's Stationery Office in 1949[17]. This study deals with the effects of the contamination produced in the Fort William area by fluorine compounds emanating from aluminum manufacturing plants. This study has been referred to as "The Fort William Study" (see also paragraphs 108 and 136).

150. Before proceeding with the more specific considerations of the effects of fluorides on human health, the Committee wishes to point out at this time that it is indeed difficult to establish "normal" values of compounds in air, water, soil, foods, etc., which in higher quantities could be hazardous. It is difficult, too, to establish "acceptable" levels of concentration of various "pollutants," recognizing that such "levels" may be changed from time to time as new knowledge warrants upward or downward revisions. However, levels or values within certain ranges are usaully adequate for the purpose of comparison and are, in most instances, acceptable as biologically significant standards.

151. The Committee wishes to point out, too, that it is difficult to compare various units of measurement as used in different situations by different workers. For instance, the concentration of fluoride in water or urine is usually expressed as so many parts per million, and is usually corrected to a specific gravity of 1.030. Other workers may express the concentration in fluids (water, urine, etc.) as so many milligrams per litre (uncorrected for specific gravity variations). The concentration of pollutants in air is expressed in several ways when dealing with various situations, e.g.: "0.69 grams of soluble fluoride per 100 square meters per month"; "0.15 micrograms per cubic meter of air"; "the amount of fluoride absorbed was 2.5 milligrams per day"; "micrograms of fluoride per 100 square centimeters per 30 days." The Committee would hope that, at some time, some international group of scientists would standardize, where possible, the units for expressing concentrations of pollutants in air, liquids and in vegetation, etc. We do recognize the several factors which make such standardization difficult or perhaps impossible; an attempt at resolving this layman's dilemma might reveal some interesting possibilities.

C. ACTIVITIES OF THE DEPARTMENT OF HEALTH IN THE PORT MAITLAND AREA — RESUME

(a) Control Legislation

152. In Ontario, prior to 1958, legislation to control smoke pollution (from combustion sources) was contained in the *Municipal Act* which allowed any municipality to pass by-laws for the control of smoke emissions. Since enforcement of legislation was a municipal responsibility, there was little uniformity in air pollution control activity in the province.

153. In 1958, the *Air Pollution Control Act* was passed which enabled municipalities to develop by-laws to control all types of air pollution. Again, this was a municipal concern and the Province acted only in an advisory capacity. In 1963, the *Air Pollution Control Act* was amended to provide for approval by the Province of new industrial processes where potential air pollution problems might be evident prior to the construction of that plant. The amendment also applied to existing industrial sources which were to undergo alterations and/or modifications in their processes. (The actual control of emissions remained with the municipalities.)

154. Finally, in 1967, the Province introduced a new *Air Pollution Control Act* which provided for the total control of air pollution by the Province, the Act to be administered by the Department of Health. The Act covers the approval for new and existing industrial operations, the control of air pollution at the source, and provides as well for the control of air pollution sources which were previously exempted in the 1958 Act.

155. The new Act (1967) also provides for the investigation of suspected cases of economic loss due to air pollution injury or damage to livestock, crops, trees or other vegetation. Should such economic loss be confirmed, provision has been made to provide a Board of Negotiation if the claimant and the owner or operator of the pollutant source are unable to arrive at a prior settlement. This provision does not preclude legal action, should settlement not be reached by negotiation. Damage alleged to be caused by sulphur fumes is still assessed and negotiated by the Sulphur Fumes Arbitrator, Department of Mines.

(b) Human Health

156. Since the Department of Health, through one or more of its branches or services, has been directly involved in studies carried out in the Port Maitland area, the degree and extent of their

activities must be indicated. This may best be done by recording their involvement in a chronological manner. A brief summary of this has already been given in Chapter I, in order to indicate the general nature of the problem.

157. In the Province of Ontario, the health of the local residents in a community is under the general jurisdiction of the Medical Officer of Health. The Medical Officer of Health is appointed by a local Board of Health. The Provincial Department of Health acts in an advisory and consultant capacity to the Medical Officer of Health in matters concerning the health of the residents in the community.

158. The health of the workers in industrial plants is under the jurisdiction of the Department of Labour. The Department of Health has no specific legislation covering workers in an industrial plant. The Department of Health acts only in an advisory and consultant capacity to the Department of Labour in the field of industrial health. It plays a similar role to the Department of Mines in mining and metallurgical operations.

159. The Department of Health has maintained a specialized Occupational Health Laboratory service for many years. This laboratory is distinct from the Radiation Protection Laboratory and the laboratory of the Air Pollution Control Service. This latter laboratory began operation in 1959. It is the policy of the Department of Health to report the results of all tests made on human specimens, e.g., blood, urine, etc., only to physicians. This is regarded as confidential medical information requiring medical interpretation. The results of other tests, e.g., water, are sent to the Medical Officer of Health or to the individual, depending on the circumstances.

160. In 1960, when the ERCO plant was built at Port Maitland, the Air Pollution Control programme and the Industrial Hygiene programme were both in the Industrial Hygiene Branch of the Department of Health. In July, 1966, this Branch became the Environmental Health Branch, and Air Pollution Control Service and Occupational Health Service were established as units within the Branch.

161. On July 20th, 1959, a meeting of representatives of ERCO, the Department of Labour, and the Department of Health was held. Mr. H. M. Nelson, Senior Engineer, Industrial Hygiene Branch, was the representative of the Department of Health. Plans were reviewed for the new plant which was to be built to produce phosphoric acid and inorganic phosphate at the Port Maitland site.

It should be pointed out that the Dominion Fertilizer plant was first built in this area in 1958 and was originally designed for the production of single superphosphate fertilizer. In 1959, this company became a wholly-owned subsidiary of the Electric Reduction Company (parent company Albright and Wilson, England). The plant was converted to produce triple superphosphate as well as the single superphosphate. The plant to produce wet-process phosphoric acid was built in 1961 and the new plant began operation in July, 1961.

162. Subsequently, on October 18th, 1962, an inspection was made of the ERCO plant by Mr. H. M. Nelson of the Department of Health, and Mr. F. Brisco, Industrial Safety Officer, Department of Labour. Mr. Nelson commented at the time of this visit that the working conditions in general were satisfactory but some dusty areas around the fertilizer crushing area were noted. The adequacy of the ventilation provided in the plant was reviewed at the time of this visit. Mr. Nelson recommended that air sampling for fluoride should be carried out by the Industrial Hygiene Laboratory in about six weeks time, when cold weather had set in and consequently doors and windows in the plant would be closed.

163. Air sampling for fluoride was done by the Industrial Hygiene Laboratory on December 5th, 1962. Results of the sampling showed high fluoride levels in the fertilizer plant, especially around the den and in the storage area.

164. It should be mentioned that in-plant air sampling is carried out periodically by the Industrial Hygiene Group as part of the routine surveillance programme where any potential hazardous material is used, such as lead, mercury, silica, asbestos, and fluoride.

165. Air sampling provides a guide in the control of health hazards in a plant. Threshold Limit Values (T.L.V.) are used in Ontario as guides only and should not be regarded as fine lines between safe and dangerous concentrations. T.L.V.'s refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse effects. The values are time-weighted average concentrations for an 8-hour work day. The T.L.V. for hydrogen fluoride gas is 2 mg. per cubic meter of air; for particulate fluorides it is 2.5 mg. per cubic meter of air. The T.L.V.'s are set by the American Conference of Governmental Industrial Hygienists. They are reviewed annually. Dr. E. Mastromatteo, of the Ontario Department of Health, is the Canadian representative on this Committee.

166. A further plant inspection was made on February 5th, 1963, by Mr. H. M. Nelson, Industrial Hygiene Branch, and Mr. R. Greenop, Ontario Department of Labour, to check on the recommended improvements on ventilation in the plant and to attempt further air sampling for fluoride.

167. During the year 1963, approximately 20 water samples were collected by the Air Pollution Control Section of the Industrial Hygiene Branch, in the Port Maitland area, for fluoride analysis. The water samples were taken from wells, cisterns and ponds. The fluoride results ranged from 0.2 p.p.m. to 4 p.p.m.

168. On September 17th, 1965, a letter was sent to the Department of Health from Dr. W. C. B. Mills, Medical Officer of Health for Sherbrooke and Moulton Townships. The letter stated that some cattle in the Township of Moulton were affected by fluoride poisoning. Dr. Mills requested information as to whether these cattle were obtaining fluoride from pasture grazing land, and it was believed this fluoride was coming from a nearby plant.

169. Dr. E. Mastromatteo, who at that time was the Assistant Director, Industrial Hygiene Branch, and Dr. V. L. Tidey visited the Port Maitland area on October 22nd, 1965. A tour of the Electric Reduction Company plant was made with Mr. D. W. Bertram, Process Investigation and Control Superintendent. It was requested at this time that samples of urine be submitted to the Industrial Hygiene Laboratory from workers in the high fluoride hazard areas.

170. Following the plant inspection, a tour was made of the surrounding farm area and a visit was made to the farm of Mr. T. Zynomirski. Some damage was noted to trees and vegetation in the area. Several vegetation samples were obtained and submitted to the laboratory for fluoride analysis.

171. Following the inspection, a meeting was held with Dr. W. C. B. Mills, Medical Officer of Health, at his Dunnville office. The air pollution problem as it related to humans was discussed at this time. Dr. Mills had raised the question concerning the milk from cattle grazing in this area. Based on the assessment of medical literature, it was felt that there would not be significant fluoride in the milk. Dr. Mills was not aware of any health problems involving local residents at this time.

172. Following the inspection, urine samples from workers in high hazard areas at the ERCO plant were submitted to the Industrial Hygiene Laboratory for fluoride analysis. In keeping with the policy of the Department of Health, the results of the urine

analysis were sent to Dr. W. C. B. Mills, Dunnville, Ontario. The results ranged from 0.2 to 6.2 mg/litre fluoride (approximately 0.2 to 6.2 p.p.m. F).

173. Incidentally, as well as the air sampling programme mentioned above, the Occupational Health Service also carries out many health monitoring programmes, on a continuing basis, for workers where potentially hazardous materials are used, e.g., lead, mercury, silica, asbestos, benzol, and fluoride. The complete programme consists of periodic inspection of the plant by an industrial physician or an industrial hygiene engineer, periodic air sampling, and the submission of specimens from workers to the Industrial Hygiene Laboratory for analysis. As an example, in a high lead exposure area, urine samples are required once a month from every worker in the hazard area.

174. On October 20th, 1966, a complaint was sent to the Department of Health from the United Steelworkers of America about conditions in the calcium phosphate building in the Electric Reduction plant, Port Maitland. The letter stated that there was excessive dust and fumes in this area. The complaint was investigated by the Department of Labour and the Department of Health. Mr. H. Nelson, Senior Engineer, inspected the plant for the Department of Health.

175. On July 19th, 1967, Mr. A. Rayner, Chemist, Air Pollution Control Service, while on an inspection tour of the Port Maitland area, checking on agricultural injury, visited the Casina farm, at R.R. #6, Dunnville. It was reported that Mr. Joe Casina, Sr., was hobbling about on crutches because of swollen ankles and insteps. Mr. Casina felt that his condition was due to fluorosis. This was the first alleged case of human fluorosis reported to the Department of Health.

176. On July 20th, 1967, Dr. V. Tidey, Occupational Health Service, visited the Port Maitland area to investigate the claim of Mr. Joe Casina, Sr., that he was suffering from fluorosis. He met Dr. C. B. Greene, Dunnville, Mr. Casina's physician, and discussed the case with him. Dr. Greene stated that Mr. Casina had been seen in consultation recently by Dr. F. G. Marson, Hamilton, a specialist in internal medicine. Mr. Casina had been told by his attending physician that there was no evidence of fluorosis. Mr. Casina's x-rays were reviewed at the Haldimand War Memorial Hospital, Dunnville.

177. Mr. Joe Casina, Sr., was then visited by Dr. Tidey at his farm; the medical findings were discussed with him and samples

of blood and urine were obtained and submitted to the Industrial Hygiene Laboratory. The urine was reported containing 0.6 mg/l F and the blood as 0.017 mg/100 ml. F. These tests were well within the normal limits. A letter was sent by Dr. Tidey to Mr. Casina on August 2nd, 1967. Mr. Casina was told in this letter that the tests were well within normal limits. A water sample was not taken at the time of this visit because Mr. Casina had been drinking town water since 1964.

178. On the same day, at the suggestion of Mr. Casina, a visit was made by Dr. Tidey to Mr. Ted Boorsma's farm. There were three cows in the barn at this time with deformed joints and mouth "disease." It was stated that the "disease" was fluorosis. An adjacent field of clover was badly burned. Several trees, shrubs and areas of the garden were also burned.

179. Dr. F. G. Marson, Hamilton, was contacted by Dr. Tidey following this visit. Dr. Marson had been the chief consultant for all of the Dunnville doctors for about nine years. He had been working constantly with all of the doctors and was well aware of the alleged livestock and crop damage due to air pollution in the area. Dr. Marson had done much research into the literature on human fluorosis. Mr. Casina had been told that he was not suffering from fluorosis.

180. Later, we learned that Mr. J. Vanderbeek from the Port Maitland area had been in Chedoke Hospital, Hamilton, in 1965, for investigation by Dr. Marson. This man was very concerned about fluorosis at that time. All of the tests were normal and there was no evidence of fluorosis.

181. During the months of February, July and September, 1967, several samples of water were collected in the Port Maitland area and tested for fluoride at the Air Pollution laboratory. These samples included well water, deep well water, and open pond water. The results were between 0.2 and 2.0 p.p.m. fluoride.

182. On July 25th, 1967, Mr. J. Casina sent Dr. Tidey a copy of a water analysis report from the Ontario Water Resources Commission. The report contained the results of fluoride analysis of a number of water samples apparently taken in November and December, 1965, in Sherbrooke Township. The report had been sent to Dr. W. C. B. Mills, Medical Officer of Health, Township of Sherbrooke, from the OWRC in 1965. The first time that this report was brought to the attention of the Environmental Health Branch was on July 25th, 1967. And incidentally, but remarkably, this

report was provided to the Branch by Mr. J. Casina, Sr., who had had the report for some months!

183. As a result of the public interest generated by the CBC television programme "Air of Death," shown on October 22nd, 1967, in which it was reported that persons living in the "Dunnville" area were suffering from fluorosis, the Department of Health undertook a complete environmental health survey in the area.

184. The study included the following:

1. General features of the area.
2. Air sampling for gaseous and particulate fluorides.
3. Water sampling.
4. Soil.
5. Vegetation.
6. Livestock.
7. Wildlife.
8. Humans.

185. This investigation by the Department of Health, of human fluorosis in the area, covered two main aspects—a health survey of the residents of the area and studies on in-plant workers at ERCO. These two studies are particularly relevant to the inquiry and are reported at this time.

186. The health survey (community study) of the residents in the Port Maitland area was made by Dr. V. Tidey, Occupational Health Service, in November and December, 1967. He was assisted by Miss E. Peckham, Industrial Health Nurse, Miss M. Waldon, Public Health Nurse, and Mr. D. Bogaerts, Public Health Inspector. The area covered by the survey encompassed the ERCO plant and extended one-half mile up-wind and nearly five miles down-wind from the plant. At each home in the area the following procedure was carried out:

(a) A health questionnaire was filled in by a Department official to include the name, age, and school of each child, health complaints, name of family physician, dentist, and an occupational history.

(b) A urine sample was obtained from one member of each residence.

(c) A sample of the drinking water was obtained from each home.

The urine and water samples were submitted to the Industrial Hygiene Laboratory for fluoride analysis.

187. At each residence, it was explained to the family that the Government was offering a complete medical examination, to include blood and urine samples, to any resident in this area who might be concerned about his health in relation to the air pollution programme. The medical examination was to be made by the family physician. Following this examination, if there was any concern on the part of the patient or the physician, or if there were suggestive findings of fluorosis, those persons would be offered the opportunity for a consultation with Dr. K. J. R. Wightman, Professor of Medicine at the University of Toronto. Hospitalization would be provided where indicated. The cost of medical investigations would be borne by the Government.

188. In the health survey, 195 families were contacted and health information was obtained on over 700 people. From this survey, it was estimated that about 130 people would be going to their family physician for a complete medical examination. However, by the end of March, 1968, only 45 residents had taken advantage of the opportunity of having this special examination. A total of 6 persons was referred to Dr. K. J. R. Wightman for further study.

189. A dental survey was made of the children in the area by Dr. Ralph C. Burgess, Faculty of Dentistry, University of Toronto, Toronto, Ontario. The results of this study are reported later in this chapter.

190. A report on the health survey was submitted to the Committee of Inquiry at the public hearing in January, 1968, at Cayuga, Ontario. Following the public hearings, the results of the urine analyses were sent to the family physicians in the area.

191. An in-plant study of the employees of the Electric Reduction Company at Port Maitland was made in November and December, 1967, by Dr. G. B. Nelson, Occupational Health Service. The examination of each worker included:

(a) A detailed occupational history.

(b) A detailed functional inquiry.

(c) A general physical examination.

(d) Blood and urine fluoride determination.

(e) X-rays, if indicated.

192. Criteria used for the diagnosis of fluorosis by the Department of Health were:

(a) A history of prolonged occupational exposure.

(b) Appropriate symptoms and findings.

(c) Elevated excretion of fluoride in the urine.

(d) X-ray changes compatible with the diagnosis.

These criteria were selected after an exhaustive study had been made of the scientific literature relevant to human fluorosis.

D. AIR POLLUTION MONITORING PROGRAMME

(a) Local Area

193. As prviously mentioned, the Dominion Fertilizer Company's plant at Port Maitland was purchased by ERCO in 1959 and expanded in 1961 to include the manufacture of phosphoric acid and triple superphosphate fertilizer. In 1960, the Air Pollution Control Service of the Department of Health set out "limed" candles so that the relative fluoride concentrations in the atmosphere could be determined. "Lead peroxide" candles were at the same time set out to obtain information on the sulphur dioxide (SO_2) levels relative to emissions from the operating Sherbrooke Metallurgical Company plant. The area covered by the sampling extended some five miles from the plants. The original number of lime candles was increased in March, 1962, from 11 to 16 and to 17 in September, 1963. The lead-peroxide candles were increased from 6 to 11 in March, 1962, and to 12 in September, 1963. These "candles" consist of chemically treated paper or cloth of a standard size (100 square centimeters) which are fastened to a cylindrical form. The prepared form is secured in a louvered, protective shelter and exposed to the atmosphere for a given period of time (30 days). At the end of a sampling period, the exposed surface is removed and analyzed for accumulation of either "fluorides" or "sulphur dioxide." This technique enabled the Air Pollution Control Service to measure relative amounts of either substance in an area over a 30-day period.

194. The levels of fluoride concentration remained relatively constant and "safe" (under 100) for several years. Early in 1965, there was a sudden and dramatic increase in the fluoride readings (Table V).

195. A few figures, taken from Table V, are brought together in Table VI, so that the magnitude of the increases in fluoride concentrations that took place in 1965 may be emphasized. An examination of ERCO's production figures for these years does not reveal any information that could account in itself for the 1965 increases.

TABLE V

AIR SAMPLING DATA, PORT MAITLAND AREA
Limed Candle Method—Measure for Fluoride
Micrograms F/100 cm²/30 Days

	Annual Mean Levels—12 months				*Mean Levels—Growing Season, May through September*			
Station	1964	1965	1966	1967	1964	1965	1966	1967
A	80	3642	2596	1325	106	2260	2210	946
B	66	987	601	401	35	1385	702	397
C	54	2008	1090	560	40	978	733	268
D	61	1553	1280	510	25	638	634	211
E	77	1328	715	318	49	396	402	186
F	75	809	388	182	62	984	386	169
G	82	1064	688	367	71	472	449	552
H	72	284	205	89	68	259	132	66
I	48	77	53	40	36	72	58	40
J	52	73	40	44	38	57	51	51
K	46	65	75	51	30	66	78	64
L	62	300	246	96	60	243	123	78
M	67	189	139	72	70	195	188	81
N	38	109	68	51	19	135	64	51
O	39	95	43	34	86	48	39	—
P	29	41	42	28	23	33	25	23

Lead Peroxide Candle—Measure for Sulphur Dioxide
Milligrams SO_3/100cm²/Day*

	Annual Mean Levels—12 months				*Mean Levels—Growing Season, May through September*			
Station	1964	1965	1966	1967	1964	1965	1966	1967
1	1.47	1.40	1.34	1.17	1.08	0.89	1.00	0.75
2	0.75	0.71	0.64	0.54	0.67	0.69	0.58	0.50
3	1.30	1.66	1.40	1.14	0.47	0.61	0.49	0.45
4	1.26	1.51	1.49	0.94	0.53	0.65	0.66	0.47
5	1.07	1.16	1.04	0.86	0.65	0.67	0.54	0.55
6	1.06	1.07	0.80	0.75	0.78	0.98	0.74	0.65
7	0.62	0.81	0.73	0.53	0.51	0.64	0.52	0.46
8	0.58	0.72	0.69	0.56	0.46	0.71	0.53	0.47
9	0.42	0.53	0.48	0.44	0.26	0.45	0.31	0.36
10	0.41	0.48	0.49	0.40	0.26	0.39	0.27	0.31
11	0.31	0.39	0.35	0.32	0.03	0.26	0.22	0.24

*Accepted analytical technique used in reporting sulphur compounds to indicate presence of gaseous sulphur compounds.

TABLE VI

Mean Levels for 12 months expressed
as Micrograms F/100 cm²/30 days

Station	1964	1965	1966	1967
A	80	3642	2596	1325
B	66	987	601	401
C	82	1064	668	367
D	67	189	139	72

196. Since the "candle method" indicates relative amounts of fluoride over a given period of time, it is difficult to interpret any single reading in a valid way. There are major variations in monthly results at any individual station which may be attributed to plant production, wind direction and other meteorological conditions. Therefore it is necessary to assess this data on the basis of long-term averages rather than as single readings, since significant "pollution" damage is the result of continuous rather than sporadic atmospheric pollution. In order to assess long-term levels of both fluoride and sulphur dioxide in the Port Maitland area, Mr. D. Bogaerts, Chief Inspector, Pesticides Control, Ontario Department of Health, prepared, at the request of the Committee, a series of maps and "overlays" which represent graphically the levels of fluoride and sulphur dioxide for the years 1965, 1966, and 1967. Figure 4 shows a series of semi-circles which enclose a given area. These "boundaries" are called "isopleths," a graph which plots the existence of a given phenomenon in meteorology. In this instance, the isopleths indicate the varying amounts of fluoride in the atmosphere within the defined area*. By plotting the annual mean levels of fluoride of each station (16 in all), it is possible to "enclose" an area that has a reading of over 100 or 200 micrograms fluoride per 100 square centimeters. The map overlay for the year 1965 shows isopleths of 100 reaching to a 6-mile radius, e.g., Stations M and N. Readings of over 200 cover areas as far as four miles from the plant complex. It is significant to note the conformity of the isopleths with the area covered by the polar plot previously mentioned (see paragraph 10). The high F levels fall within the "cone" area determined by prevailing meteorological conditions (see Figure 4).

197. In 1966, areas which gave values of 100 micrograms or more per 100 square centimeters were confined to a four-mile radius

*The research of Dr. E. R. Hendrickson in Central Florida, where there is a large concentration of fluoride-emitting plants, indicated that readings of 100 and 200 micrograms per 100 square centimeters indicate potential difficulties for cattle and citrus vegetation.


Port Maitland
Pollution Study
FIG. 4
LIMED-PAPER CANDLE
RESULTS 1965 AND 1967
AREAS ENCLOSED BY ISOPLETHS
100 AND 200 MG. F/100 CM²/30 DAYS-1965
AREAS ENCLOSED BY ISOPLETHS 300 AND 400
MG F/100 CM²/30 DAYS-1967.
N
S
1
2
3
4
5
100
200
300
400
Franconia
FORKS ROAD
DUNNVILLE
CANADIAN NATIONAL RAILWAY
KELLY
Broad Cr.
Disused Feeder To Old Welland Canal
Stromness
Port Maitland
DUNNVILLE AERODROME
Byng
11
40
39
Connor Bay
Splatt Bay
Mohawk Bay
Rockhouse Point
Molton Bay

and to a narrower strip of land. Readings of 200 micrograms were reduced to the three-mile radius and confined to an even more narrow area (see Figure 4). By the end of 1967, the readings of 100 micrograms were reduced and narrowed to the 2½ mile radius while the 200 microgram readings were reduced to the 1¾ mile radius and along a more narrow corridor.

198. It should be noted here that, early in 1966, ERCO installed major control devices (two new scrubbing units costing $550,000) which were aimed at reducing total fluoride emissions into the atmosphere. The noted decrease in fluoride levels in the surrounding area in the years 1966 and 1967 reflects the installation of this additional air pollution control equipment as well as periodic cutbacks in plant production. (We are unable to decide which was the greater factor.) The "limed" candle results for 1967 were approaching the 1962-1963 levels in the immediate vicinity of ERCO and within two miles down-wind.

199. Since "limed" candles can give only a relative measure of atmospheric fluorides over a given period of time, and since day-to-day measuring by this method is impossible, the Department of Health, in 1964, commissioned the Ontario Research Foundation to design an automated fluoride analyzer.

200. The difficulties in devising such instruments for automatic and continuous quantitative estimations of atmospheric fluorides may be imagined, since it required three years for the ORF to develop and build the first unit, which was installed in June, 1967. The second and third units were installed in October and November, respectively. The results obtained from these new samplers, which unfortunately cannot be compared directly with results obtained from the "limed" candles, showed a decrease of some 90 per cent in the atmospheric fluoride during the period July to September, 1967. Unfortunately, results from the automatic analyzers are expressed as micrograms of fluoride per cubic metre, F/m^3 ($1\ \mu g\ F/m^3 = 1.20$ p.p.b. at 20°C), while limed candle results are expressed as $\mu g\ F/100\ cm^2/30$ days. Since the automatic analyzer results reflect atmospheric fluoride at a given point in time, and "limed" candles show a cumulative amount of F over 30 days, it has not been scientifically possible to compare the analyzer results with "lime" candle results. Both techniques do, however, show declining amounts of fluoride in the atmosphere in the Port Maitland area during 1966 and 1967.

201. As has been mentioned briefly in paragraph 31, an independent industrial pollution consulting firm from the U.S.A., Re-

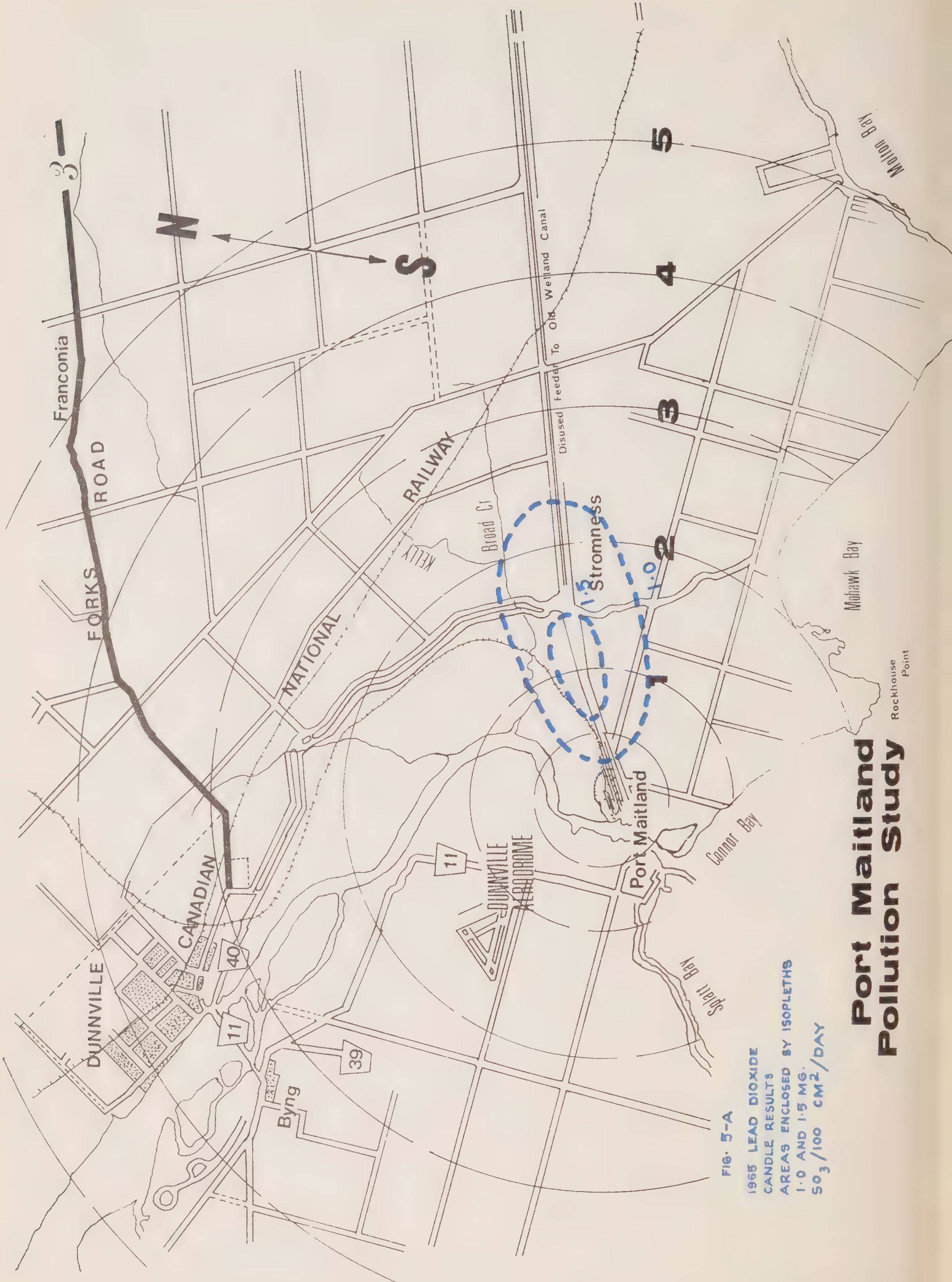


FIG. 5-A
1965 LEAD DIOXIDE
CANDLE RESULTS
AREAS ENCLOSED BY ISOPLETHS
1.0 AND 1.5 MG.
SO_3/100 CM^2/DAY

sources Research Inc., was employed by the Department of Health, in 1967, to assess and test the air pollution control equipment as installed and used by ERCO. Their tests indicated a reduction in total emissions of fluorides of approximately 90 per cent as compared to the calculated fluoride emissions of mid 1965. Although ERCO's progress in reducing fluoride emissions from its plant was substantial, nevertheless, in July, 1967, to reduce further the emissions, it was necessary also to reduce production to 50 per cent and, on October 1st, a further reduction in production to 25 per cent had to be made. The continuous monitoring units substantiated the reduction in production in the period July to October.

202. The 11 "lead peroxide" candles, used to measure sulphur dioxide in the atmosphere, did not show appreciable changes from 1962 through 1967 (Table V). The concentration of SO_2 in the atmosphere for the years 1965 through 1967 are shown in Figures 5-A and 5-B. Isopleths have been plotted on the basis of these data to show levels of 1.0 and 1.5 mg SO_3/100 cm^2/day (Figures 5-A and 5-B).

203. It was mentioned that efforts had been made by ERCO from 1960 through 1965 to control, more adequately, emissions from and in the plant. That dust and fume controls were still inadequate in 1966, in spite of the installation of additional scrubbers and ventilation equipment, is revealed through the letter received from the United Steelworkers of America (October 20th, 1966) by the Occupational Health Service of the Department of Health. This has been referred to in paragraph 174. This complaint was investigated by the Department of Labour, presumably on February 1st, 1967, when Mr. Nelson of the Occupational Health Service visited the plant with a Department of Labour representative.

204. Air sampling was carried out within the plant on November 15th, 1967, with the following results:

IN-PLANT AIR SAMPLING DATA

Location	*mg/cu. m. (as HF)*
Acidulation area, #1	0.35
Acidulation area, #2	0.34
Filtration (H_3PO_4) area, #1	0.07
Filtration (H_3PO_4) area, #2	0.06
Den (ground floor near control panel)	1.90
Den (reaction floor, in fumes)	16.10
Den (reaction floor, control room)	0.92
Fertilizer storage (catwalk over piles)	1.67
Pug mill (not operating)	0.04

FIG. 5-B
1967 LEAD DIOXIDE
CANDLE RESULTS
AREA ENCLOSED BY ISOPLETHS
1.0 MG. SO_3/100 CM^2/DAY

205. On January 28th, 1968, air samples taken from the den and storage area (where suitable respiratory protective equipment is supplied to the men) gave the following results:

AIR SAMPLING DATA—DEN, STORAGE AREAS

Location	*mg/cu. m. (as HF)*
Den (ground floor)	19.0
Den (reaction floor)	86.0
Den (control room)	7.3
Catwalk over triple superphosphate	5.96

These are the high risk areas in the plant.

E. AREA STUDIES

(a) Urinary Fluorides of Plant Workers

206. As indicated in the résumé of the activities of the Department of Health, part of this study was the medical examination of the workers in the ERCO plant. Accordingly, a medical evaluation was carried out by Dr. G. B. Neeson of the Occupational Health Service over a six-week period in November and December, 1967. The health of the in-plant workers was assessed on the basis of an adequate questionnaire and a comprehensive medical examination wherein the Department of Health's criteria were used. Urinary fluoride levels were determined and, in two cases, x-rays of the low back area were also taken. The workers were classified as Risk Groups I, II, and III, with Risk Group I having the highest overall exposure to fluorides. Since many of the men work in more than one area of the plant in the course of the day the determination of the grouping was understandably difficult. The classification was arranged by Dr. Neeson, in conjunction with ERCO officials, with reference to the available data regarding air levels of fluoride, the percentage of time spent in high level areas, the length of time on the job, and personal judgment. The urinary fluoride results correlated reasonably well with the classification made and it appeared, therefore, that such analyses would be adequate for the purpose of exposure control.

207. Urine samples for a 24-hour period collected from ten workmen who were classified as being in the high exposure area (Risk Group I) and these samples were analyzed for fluoride concentration. The results are presented in Table VII.

TABLE VII

URINARY LEVELS OF HIGH RISK WORKERS

Employee Code No.	*Sp. Gr.*	*Volume in c.c.*	*F mg/l*	*F mg/24 hr.*
1-17	1.022	825	3.6	2.96
1-18	1.018	2370	2.6	6.2
1-19	1.026	930	6.4	5.95
1-21	0.014	1240	2.9	3.7
1-22	1.020	1160	3.1	3.7
1-25	1.024	775	4.8	3.7
1-30	1.016	980	6.6	6.4
1-31	1.018	880	8.5	7.5
1-33	1.018	815	6.8	5.5
2-1	1.020	2080	0.9	1.9

208. Urine samples were collected from 260 plant workers in the three Risk Groups, including the ten in Risk Group I above, and fluoride analyses made. The results are presented in Table VIII.

TABLE VIII

URINARY LEVELS OF 260 PLANT WORKERS

Group	*p.p.m. less than 1*	*p.p.m. 1-2*	*p.p.m. 2.1-3*	*p.p.m. 3.1-4*	*p.p.m. more than 4.1*	*Totals*
I	15	6	7	4	5	37
II	46	18	2	4	3	73
III	107	35	8	0	0	150
	168	59	17	8	8	260

It is to be noted that, of the 260 workmen, eight showed a urinary fluoride concentration in excess of 4 mg/litre (specific gravity uncorrected to 1.030). Of these eight men, five were in Risk Group I. These five, together with one from Risk Group II (Employee Code Number 2-1), were among the ten persons whose 24-hour urinary fluorides are reported in Table VII. It will be noted that the values varied from 1.9 to 7.5 mg. F in the 24-hour sample. The average level of 4.76 mg. F is considerably below the mean of 9.03 mg. F per 24 hours, as found in the greatest risk group in the Fort William Study. There, in that group, the range was from 2 to more than 16 mg. F. None of those exposed Fort William workmen was found to suffer clinical disability, although in some of the older workers bone changes were noted by x-ray examination.

209. We are convinced that urinary fluoride values of up to 4.0 mg/l F are within normal limits and we are fairly sure that excretion of up to 8 mg/l F over prolonged periods of time is not indicative, in itself, of chronic fluorosis in humans. Even so, and even with the evidence that only 3.2 per cent of the ERCO plant workers showed urinary values in excess of 4.0 mg/l and none above 7.5 mg. per 24-hour sample, and in spite of the fact that no clinical or x-ray evidence showed that any of the workmen had chronic fluorosis, we urge ERCO to take appropriate steps to ensure that 24-hour urinary fluoride determinations are made on an annual basis of all workmen in the Risk Group I. We urge, too, that more adequate in-plant air sampling be carried out at appropriate intervals. We feel that a physician, even on a part-time basis, should have been on ERCO's staff as early as 1960. The company has a specific responsibility to ensure a health hazard-free working environment for its employees. It is fortunate that the levels of fluoride and the length of exposure in the risk areas were such that no human fluorosis developed.

210. We are convinced that none of the workers at the ERCO plant showed any evidence of human fluorosis. Nevertheless, ERCO should maintain a vigilant concern for the long-term health of its workers.

(b) Urinary Fluorides of Area Residents

211. As part of the Department of Health's plan to conduct a complete environmental health survey of the area near the two industrial plants, urine specimens were obtained from 213 area residents and analyzed for fluoride. The so-called "raw" results were made available to the Committee. Of these, 119 showed 0.2 p.p.m. or less of fluoride, 71 ranged between 0.3 and 1 p.p.m., and 20 showed more than 1 p.p.m. of fluoride in their urine. In this group of 20, the 4 highest values recorded were 2.2, 2.4, 2.6, and 3.4 p.p.m. of fluoride. As a total group, these values are particularly low, but the latter four individuals, even though their urinary fluorides are within normal limits, should be checked further (see paragraph 208). There is, however, no suggestion that these persons had been ingesting any abnormal amounts of fluoride.

F. DENTAL SURVEY OF AREA SCHOOL CHILDREN

212. So far, it has been established that there was no evidence of fluorosis in any of the in-plant workers. It has been shown, too, that the drinking water consumed by residents of the area did not contain sufficient concentrations of fluoride to produce any signs or

symptoms of chronic fluorosis. It was considered essential, nevertheless, to have a dental survey conducted on school children in the area. This study was commissioned, and was undertaken by Professor R. C. Burgess and Professor D. W. Lewis of the Faculty of Dentistry, University of Toronto. Under adequate lighting conditions, the degree of "mottling" of the area children's teeth was determined. The survey would demonstrate the fluoride intake of the children prior to 1965: such dental examinations were conducted on 184 school children in the area. Because of its relevance, Burgess and Lewis' report is presented here in its entirety:

213. "Teeth are susceptible to 'mottling' from excessive fluoride intakes during the period when the organic matrix of the enamel is being formed. Different teeth are in this susceptible phase of development at different periods of childhood: permanent incisors, first bicuspids and first molars (Group 1 teeth)—between 1 and 4 years of age; permanent cuspids, second bicuspids and second molars (Group II teeth)—between 3 and 7 years of age. Deciduous teeth are never noticeably mottled, presumably because their enamel matrix is formed mainly *in utero* where the passage of fluoride from maternal to foetal blood is restricted by the placenta (Zipkin and Babeaux, 1965)[30].

214. "Since 'mottling' defects are permanent, they provide a visible record of the individual's fluoride intake during childhood. A large increase in fluoride intake of a group of children is dramatically recorded in the permanent teeth which are being formed at that time—this record is revealed several years later when the teeth erupt into the mouth (Kempf and McKay, 1930)[31].

215. "The severity of fluoride mottling ranges from barely detectable white lines or patches in the enamel (very mild) to a confluent pitting and brown staining of most of the enamel (severe). The criteria for each category have been described by Dean[32].

216. "In North America, food contributes a small and rather constant amount of fluoride to the total daily fluoride intake. McClure (1938)[33] states this is due to the fact that our principal foodstuffs (cereal grains, fruits, vegetables, meat, milk and eggs) are uniformly low in fluoride. The major source of difference in daily fluoride intake between population groups lies in the fluoride content of their drinking water supplies (McClure, 1943)[34]. People using water containing 1 p.p.m. and 5 p.p.m. fluoride have, on the average, a total daily fluoride intake which is three times and eleven times, respectively, that of people using low fluoride water (0.1 p.p.m. F).

217. "Several studies (Dean, Elvove, 1941)[35] have demonstrated the close relationship that exists between water fluoride content and the incidence and severity of mottled enamel. In the Northern United States and Canada, there is no aesthetically significant mottling (moderate to severe categories) when the drinking water contains less than 1.8 p.p.m. fluoride. With progressively higher fluoride intake, the incidence and severity of mottling increases proportionally (Appendix IV).

218. "The present study was undertaken to obtain some indication of the past fluoride intake of residents of the Port Maitland area. If the operation of the Electric Reduction Company plant had significantly increased the daily fluoride intake of nearby residents, this should be documented by a dramatic increase in the frequency and severity of mottling of the children's teeth. One should also be able to estimate the average daily fluoride intake from the mottling data. It should be emphasized that this 'biological assay' of fluoride intake is extremely sensitive. The use of drinking water containing 5-6 p.p.m. fluoride will produce moderate to severe mottling in almost 90 per cent of the children, whereas even higher concentrations (8 p.p.m.) can be consumed for a lifetime without otherwise affecting the general health of the individuals (Leone, 1954)[36].

219. "Our intention in this study was to determine the incidence and severity of mottling in children of the Port Maitland area and to estimate the fluoride intake responsible, in terms of p.p.m. water-borne fluoride which would produce the same degree of mottling.

220. "Dental examinations were carried out on 184 children living within a seven-mile radius of Port Maitland in the sector extending from Lake Erie to Dunnville. These children were pupils at Stromness, Sherbrooke, Lowbanks, Anna Mealick and St. Michael's Public Schools. The study group included practically all the children living within 4 miles of Port Maitland and a sample of children living from 4 to 7 miles away. Names of the latter group of children were randomly selected from the class registers.

221. "The children's teeth were examined with the aid of a Cameron-Burton intra-oral examining light, or a flashlight providing equal illumination*. Tongue depressors were used for cheek retraction and cotton gauze pads were used, when necessary, to remove food and debris from the tooth surfaces. Signs of enamel

*A convenient electric outlet was not available at the two small schools—Stromness and Sherbrooke.

hypocalcification or hypoplasia on any teeth were rated in severity according to a classification adapted from that of Dean[32], and shown in Appendix V. All examination were done by the same person (D.L.). He provided a verbal description of the teeth present and enamel abnormalities thereof to a second person (R.B.) who recorded the findings on standard dental charts. Neither person was aware of the place or duration of residence of the children at the time of examination. Data on the incidence and severity of mottled enamel in 151 of the 184 children examined are presented in Appendix VI. These results are expressed as frequency per cent (Appendix VII). Data from 33 children were not included: 8 of these children had no permanent teeth, as yet, and the other 25 children moved into the Port Maitland area after 1964, i.e., their presently erupted teeth had already passed the mottling susceptibility period.

223. "It must be emphasized that these clinical results make possible an estimate of the maximum fluoride intake of children in the Port Maitland area during the period 1957-1964. No statement can be made concerning fluoride intakes after 1964, since any teeth which are susceptible to mottling during 1965 and 1966 will not erupt into view until 1969 or 1970.

224. "The following conclusions were reached from examination of the data: The per cent frequency of the different mottling severities (Appendix VII) was very similar for Group I teeth (incisors, first bicuspids, first molars), not only in the study group as a whole, but also in the various sub-groups thereof. Because less than half of the children had Group II teeth present in their mouths, all subsequent statements refer specifically to the data for Group I teeth.

225. "In the study group of 151 children (Appendix VI), 119 showed no signs of mottled enamel; 32 children showed some degree of mottling on one or more teeth. Children with very mild, mild, moderate, and severe mottling numbered 29, 2, 1, and 0, respectively.

226. "The mottling frequency was calculated for children living different distances from Port Maitland (Appendix VII). Children living 4.0 to 6.9 miles away were included as a control group; 85.4 per cent of them had "normal" teeth and 14.6 per cent showed very mild mottling. This mottling frequency approximates that found in children who use drinking water containing 0.9 to 1.2 p.p.m. fluoride throughout childhood.

227. "There appeared to be a somewhat higher incidence of mottling in children living within two miles of Port Maitland. This group of 45 children included all three children with mild to moderate mottling and 13 out of the 29 children with very mild mottling (Appendix VI). It should be emphasized that only one of these children showed a degree of mottling (moderate) that was aesthetically significant. The per cent distribution of mottling severities in these 45 children (Appendix VII) approximates that found in individuals who used drinking water containing 1.5—1.9 p.p.m. fluoride throughout childhood.

228. "The sub-group with the highest mottling incidence was found to reside within two miles and to the NW of Port Maitland. The 41.6 per cent incidence of mottling (very mild to moderate) suggests that, during the period 1957-1964, these children, and presumably their parents as well, had a maximum fluoride intake equivalent to that obtained from drinking water containing 1.8—2.0 p.p.m. F. Medical studies in several North American communities have shown that the lifelong intake of drinking water containing up to 8.0 p.p.m. F is compatible with good health and certainly does not result in skeletal fluorosis.

229. "Finally, one must note that the fluoride intake of children living within two miles of Port Maitland was not high enough to cause disfiguring dental fluorosis. However, such undesirable defects will be evident in the teeth of many children if fluoride intakes since 1964 have exceeded the 1957-64 levels by 50 to 100 per cent. For this reason, it is recommended that children in the Port Maitland vicinity be re-examined for mottling defects in early 1970.

230. "In summary, dental examinations for mottled enamel (dental fluorosis) were carried out on 151 children who lived in the Port Maitland area. Only child showed aesthetically significant mottling (moderate category).

231. "Mild and very mild forms of mottling were detected more frequently in children living within 2 miles of Port Maitland (33.3 per cent) than in children living more than four miles away (4.6 per cent). The mottling frequencies for children living within 2 miles of Port Maitland indicate that their maximum fluoride intake during the period 1957-64 was equivalent to that derived from drinking water containing 1.5—1.9 p.p.m. fluoride. The lifelong intake of 3 or 4 times this concentration of fluoride would not impair general health or cause symptoms of skeletal fluorosis.

232. "The foregoing data and conclusions relate to fluoride intakes in the Port Maitland area during the period 1957-64. It is suggested that the dental examinations be repeated in 1970 to ascertain whether the fluoride intake of children, and presumably of adults as well, has increased since 1964."

We would like to acknowledge the generous assistance given to us by Mr. J. R. M. Peat, District School Inspector, and by Mrs. Malseed, Public Health Nurse.

G. EVIDENCE RELATIVE TO HUMAN FLUOROSIS— PUBLIC HEARINGS

233. It is not an easy task for the Committee to present the many inter-related aspects of this complex subject in either a chronological manner or on a witness-to-witness basis. We have, however, attempted to separate the evidence and the results of the many studies into some kind of reasonable and legible order. Thus, we now return to the evidence of "alleged" chronic fluorosis as obtained from the witnesses and the results of the examinations conducted by the several physicians concerned. But first, it must be pointed out that apprehension, fear of the unknown, and association of ideas, can cause misconceptions and belief in what actually is not necessarily true. Such fears, such concerns and such beliefs cannot simply be dismissed; they must be conclusively proven or disproven within the abilities of specialists to do so. Otherwise, the uncertainties will persist and psychological effects may become manifest. At the same time, it is mandatory that recognition be given to the fact that, in any group of some thousands of people spanning the normal age-group distribution of population, there will inevitably be persons suffering from cardio-vascular disease, respiratory disease, allergies, arthritis, rheumatism, kidney disease, post-accident disabilities, gastro-intestinal disturbances, and statistically almost every type of illness and ailment to which a population group is subject. This is not said to denigrate or to discredit any of the witnesses involved; it is pointed out only in a spirit of realism.

234. The Committee, in an endeavour to obtain as much evidence as possible, from as many residents as possible, relative to their feelings or convictions that they were suffering from fluorosis, invited such persons to appear as witnesses and to give evidence at the hearings. Other persons were subpoenaed by the Committee to so appear. Before presenting the specific evidence given under examination-in-chief or under cross-examination, a brief summary

of the past illnesses and other pertinent information respecting the witnesses' health is presented. The information, upon which the summaries are based, was, of course, obtained from the witnesses during the hearings.

WILLIAM WARNICK

235. William Warnick, R.R. #2, Lowbanks, residing about ½ mile NE of Port Maitland; age 57; shift mechanic working in Dunnville; resident of area since 1951; married with seven children.

236. Summary of previous medical history: In hospital on several occasions: 1935 (flu); 1944 (after effects of an "injection"); 1950 "nauseated stomach," . . . next day, after "some type of an attack," . . . next day, "some disc trouble"; 1955 (appendectomy); 1958 "check for ulcers"; 1959 "a wrenched elbow from a private home party, fooling around"; 1964 "upper teeth removed," . . . "upset stomach, nervous condition"; 1966 "run down condition."

237. Mr. Warnick saw Dr. Marshall, then Dr. Greene and Dr. Gardner, for "my neck and shoulders were very stiff"; 1967 "in hospital—I think I can say they suspected the possibility of cancer but . . . as negative." In 1967, on November 1st, he saw Dr. Marshall again, "mentioned the possibility of fluorides"; November 3rd, put in hospital "for a kidney x-ray," . . . "nothing wrong with the kidney," . . . "I was told that both blood and urine samples were okay" (for fluorides); November 12th, "Dr. Waldbott and I had a 10 or 15 minutes chat"; November 14th, "saw Dr. Victor Cecilioni of Hamilton"; November 30th, "had 19 back and shoulder x-rays . . . negative for fluorides."

238. Mr. Warnick summed up his ailments and complaints this way: "the following abnormalities have been increasingly more noticeable to me: stomach weaknesses, cramps, slight pains across the chest, tiredness, extreme sleepiness at times, shoulder stiffness . . . diarrhoea, lack of control, bowel-wise . . . extreme coughing at times, dizziness, slight head annoyances, hip stiffness, cramps in fingers and hands, variations in sight when I am driving a car . . . loss of sexual appetite [he has seven children], I have found that it only takes four or five bottles of beer to put me where it would have taken 12 pints of beer ten years ago . . . loss of wind, urination (frequent and uncontrolled), muscular pains, some marks . . . developing on my chest, loss in weight."

239. Mr. Warnick also gave evidence that a sample of his well water, in 1954, was tested by the Hamilton City Laboratories which reported "this water is bacteriologically unsafe for domestic

use unless boiled before using." Mr. Warnick, relative to further sampling, said "these are on my own well, 1960, May, 1960, my own well; they give an E-Coli count of 32.2; 1962 they give an E-Coli count of 39.0."

240. On the evidence of Dr. Greene and reports of other physicians, there were no clinical signs or symptoms associated specifically with the diagnosis of fluorosis; urinary fluoride was normal, x-ray examinations showed no bone abnormalities associated with chronic fluorosis. Mr. Warnick, although he may be suffering from one or several ailments, did not have fluorosis.

PETER DE RUITER

241. Peter De Ruiter, unemployed, former mill operator at ERCO; married; age 44; lives ¾ mile NE of Port Maitland in the Village of Stromness; previously worked for three months with an asbestos company in British Columbia; worked for three years at ERCO.

242. Summary of previous medical history: November 26th, 1966, witness "fell off a hopper. I was cleaning a hopper and I slipped and I fell on my back from approximately 10-12 feet. I had a spinal concussion and I had internal bleeding." He was in hospital for five days at that time; to Workmen's Compensation Board Hospital for physiotherapy until February 1967; was discharged and "Dr. Mustard sent me back to work"; on May 8th, 1967, "while driving a front-end loader, weighing at least 10 tons, it rolled back and I dropped my bucket to slow it down, but I hit a hump and she flipped over and I got pinned underneath" . . . "and then I got a squeezed rib cage"; "once in a while my legs seize up and I fall right on the floor"; "I have got sore joints, my feet are sore, my ankles are sore, my leg is sore and my back is sore"; "I have stomach disorders, my eyesight is going bad and I get severe headaches all the time"; "my legs seize up, my arms swell up. I have to blame it on something."

243. We feel that it is fairly obvious that the complaints of Mr. De Ruiter, who is presently in receipt of Workmen's Compensation, are due to his two serious accidents. There was no evidence even to suggest that "fluoride" pollution was a factor in his case.

MRS. SHIRLEY NIE

244. Mrs. Shirley Nie, housewife, resides about ⅛ mile from the ERCO plant; age 45; four children.

245. Summary of previous medical history: Since 1960, "I have had this tiredness and rheumatism or something in my shoulder

and the elbow . . . it just seems to come and go"; July, 1967, "I got the sore in my eye and it all swelled up terrible and just pained me . . . thought it was a stye."—she was treated by Dr. Rigg; September, 1967, suffered from "an infection in my kidneys" which was treated effectively; November 12th, witness was asked by a member of the "Air Pollution Committee" to be examined by another doctor (Dr. Waldbott of Detroit); "when the air is heavy, that stuff from the factories just falls right down to the ground and you can't hardly see, you know, to the next place and the smell is terrible"; the effect of the dust on the children is such that they "just itch and scratch something terrible in the summer."

246. That there has been a nuisance factor as a result of the "dust" from ERCO, there is no doubt. The itchiness of the skin of the children might be due to particulate matter, but likely not to the fluoride content of the particulates. There is no evidence which would suggest that Mrs. Nie was suffering from or was being affected by fluoride.

MRS. JUNE MacKEIGAN

247. Mrs. June MacKeigan, housewife, resides on Inman Road, R.R. #6, Dunnville, on a three-acre lot, 2½ miles NE of Port Maitland; age 45; two young children; 13 years in area; husband farmed 96 acres until 1964 when farm was sold.

248. Summary of previous medical history: October, 1960, witness had a coronary thrombosis—hospitalized about six weeks; December 19th, 1960, had a gall bladder operation—hospitalized about two weeks; July 2nd, 1961, suffered a second "heart attack"—hospitalized six weeks; "in 1962, I had another coronary thrombosis"—hospitalized three to four weeks; "I had four brothers who died with coronary thrombosis"; in March, 1967, hospitalized for a "gland condition"; "then starting at the end of May, 1967, I have had trouble with my feet right up to my elbow"; x-rays taken of feet, thigh, back and shoulders; "according to Dr. Mills, according to the blood tests and the x-rays, it is arthritis"; drinking water obtained from roof run-off, highly contaminated by E-Coli; advised "to clean out our cistern and put stuff in it [chlorine?] before we could use that water again"; difficulty in breathing (at times). There was no evidence to suggest that Mrs. MacKeigan had fluorosis.

MR. A. L. FARR

249. Mr. A. L. Farr, retired farmer now doing electrical con-

tracting; residing in Township of Sherbrooke, 3½ miles SE of Port Maitland; married; two children; age 49.

250. Summary of previous medical history: worked underground as a mucker at Sudbury in the early 1940's, "good health until four or five years ago"; appendectomy in 1948; backaches about four years ago, had chiropractic treatments for his back; does not associate "backaches" with pollution; December, 1965, "had the first signs of dermatitis show up on my hands, face and feet"; saw Dr. Marshall and later Dr. McGuinnis, then Dr. Rosenthal (dermatologist in Hamilton); dermatitis controlled by "ointment"; "my eyesight—my eyes water a great deal . . . headaches daily and I have had nosebleeds ever since I have had the dermatitis"; advised by Dr. White (Eye, Ear, Nose and Throat specialist) that "changing my glasses would not help the watering of my eyes"; "he told me it was my nerves"; witness stated that his wife, who is under the care of Dr. Marshall, with "a case of chronic arthritis," asked Dr. Marshall "in the fall, after the television programme, if there was the slightest chance . . . related in any way with the pollution"—the reply from Dr. Marshall was said to be "absolutely not"; water supply is primarily from cistern supplemented "when the cisterns get low, I pump water from the beach, from Lake Erie."

251. Again there is no evidence to associate Mr. Farr's complaints with air or water pollution emanating from the Port Maitland plants.

MR. JOSEPH CASINA, SR.

252. Mr. Joseph Casina, farmer, resides about 2½ miles NE of Port Maitland, R.R. #6, Dunnville; married; one son (21); age 55.

253. Summary of previous medical history: In 1952, Mr. Casina had a "heart attack"; suffered another "heart attack" in 1964 but continued to work the farm; "tire out awful fast in recent years" (1965); "I had a stomach condition"; "x-ray for gallstones and it showed no sign of gall bladder"; was relieved of pains by returning to previous diet; in the spring of 1967, "it didn't matter what work I did I tired out awful fast"; "you have a lot of aches and pains every spring . . . when you start in farming . . . so, I disregarded it until my foot started to swell about the end of June"; saw Dr. Greene, complained about "my back that is bothering me"; "my spine that is bothering me"; "my head bothers me too"; "my ankles still bother me"; taking pills to dull the pain (since June,

1967); seen by Dr. Waldbott in September of 1967; admitted to having been examined by Dr. Greene, Dr. Marson and by Professor Wightman; "this past summer I had a considerable amount of nosebleed."

254. Dr. Greene gave evidence to the effect that when he saw Mr. Casina on July 7th, 1967, Mr. Casina "felt very strongly at that time that it was a good possibility [that he had fluorosis], that he had reason to believe that he lived in a contaminated area and that he might possibly be suffering from it." Dr. Greene referred Mr. Casina to Dr. F. G. Marson, a recognized senior consultant in Internal Medicine, of Hamilton. Dr. Greene then testified that "Dr. Marson sent me his report on examinations of the man [and] that he could find so far as he was concerned no evidence to suggest that this man [Mr. Casina, Sr.] had fluorosis." This information was relayed to Mr. Casina by Dr. Greene. Later, Mr. Casina returned to Dr. Greene, complaining of swollen and painful ankles, and the report from the radiologist, as stated by Dr. Greene, was that "he could find no definite evidence of bone pathology" and the "physical characteristics of the bone were normal for a man of his age."

255. Dr. Marson, in his testimony, stated that he had first examined Mr. Casina on July 18th, 1967, in consultation, at the request of Dr. Marshall. In Dr. Marson's notes at that time, it is stated "the patient now has various symptoms and seems to be upset by the idea that he has the same problem himself" (as the lameness in cattle). "He was walking with a stick and professed to have extreme pain in his ankles and talked about them feeling hot and that they had been swollen so that he had to increase the size of his shoes and indeed he stated that they were still swollen, which fact I could not confirm."

256. "He confesses that he has been 'nerved up' (that was his own expression) for a few years about the local fluoride problem." Dr. Marson continued in his testimony, "the ankles were not swollen to me at all, but at first almost touching them seemed to cause exquisite pain, but later, provided I engaged him in conversation, I could move them fairly freely without too much trouble. There was certainly no local heat or redness." Dr. Marson's opinion, formed and recorded at that time, was that he may have "some condition such as rheumatoid [arthritis] in his ankles or even gout." "May require such things as a serum uric acid [re gout] and rheumatoid serology."

257. The hospital records show that Mr. Casina's blood fluoride level was 0.017 mg/100ml—the normal accepted range being 0.11 to 0.45 mg/100ml. The urinary fluoride level at that time was also normal. Dr. Marson continued, "So, when I learned that the blood fluoride level was normal (x-rays had previously shown no indication of bone pathology) . . . it was my opinion that this patient could not be suffering any symptoms of fluorosis, that he did not have fluorosis, and that his symptoms, whatever they were due to, were nothing to do with fluorine exposure." "I thought in that he was in this state of extreme apprehension, it was terribly important for that patient to be reassured."

258. This lengthy examination of Mr. Casina was conducted by Dr. Marson on 18th July, 1967. On July 20th, 1967, Dr. V. Tidey of the Department of Health, who had been very active in the area for some time, visited Mr. Casina and took samples of blood and urine. Mr. Casina wrote, to Dr. Tidey, a letter under date of July 25th, 1967, in which he states: "Dr. Tidey, samples that were taken at time of your visit probably have been completed. Dr. Tidey, my intent is not to intercede between yourself or your staff, but by the integrity shown in talking to you, urine and blood analysis, will be direct and to the point either way."

259. Dr. Tidey wrote to Mr. Casina on August 2nd, 1967, stating "the blood and urine samples that I obtained from you at the time of my visit have been reported by our laboratory. Both of these tests were well within normal limits. The urine was less than 1.0 mg. fluoride per litre which is considered a normal value." Dr. Tidey also said in that letter to Mr. Casina, "Dr. C. B. Greene and Dr. G. Marson have both been contacted and your x-rays taken at the Dunnville hospital were reviewed."

260. It is evident that Mr. Casina had been advised by Dr. Greene of Dunnville, by Dr. Marson of Hamilton, and by Dr. Tidey of Toronto, that there was no evidence of fluorosis. Yet, knowing that he did not have fluorosis, or at least having been told repeatedly that he did not have fluorosis, Mr. Casina appeared on the CBC's "Air of Death" television programme and did not state these facts—facts which the CBC might have used had they desired to be factual and responsible.

261. Dr. Marson saw Mr. Casina on referral again on October 17, 1967. The patient was still complaining of pain and swelling of the ankles, and stated, as related by Dr. Marson from his notes, "that the pain radiated up the front and back of his legs to the front of his knees and was continuous and seemed to be unaffected

by position or movement or weight bearing" . . . "he was no longer in a state of apprehension or so it seemed to me." Mr. Casina complained, as Dr. Marson put it, of a "terrible headache at all times." Dr. Marson, reading from his clinical notes, continued: "the ankles looked totally unremarkable to me and I was unable to accept that they were swollen, although he insisted that they were." Dr. Marson, in replying to a question, said: "on the basis of what I ascertained myself and the investigations that were done later, and I found the results, as far as I am concerned, I am certain as I can be of anything, that there was no evidence whatsoever that any symptoms that either of these gentlemen [Mr. Casina and Mr. Vanderbeek] had, or have, were due to fluorine exposure."

262. It is only right to point out here that Mr. Casina was not the kind of witness whose veracity one automatically tends to accept. His replies to reasonable questions were ofttimes evasive, on occasions even truculent; his outlook was certainly jaundiced, if not biased; the evidence that he did not have fluorosis did not seem to be acceptable to him, even though advised by Dr. Greene, by Dr. Marson, and by Dr. Tidey. This same general attitude was evident in those portions of the hearings dealing with "livestock" and with "crops," wherein his claims, his statements and his outlook indicated that he wanted more than his "pound of flesh" regardless of the facts.

263. But we must cite Professor Wightman's evidence, following his examination of Mr. Casina in the Toronto General Hospital. Mr. Casina, on the basis of the arrangements made through the Department of Health, was referred to Professor Wightman by Dr. Greene and Dr. Marshall, and admitted to the Toronto General Hospital in mid-November, 1967. Mr. Casina remained in hospital for four weeks and, during that time, was seen by several members of the attending staff, including Dr. E. Yendt (a recognized specialist in the field of fluorosis) and Dr. McCarthy (specialist in rheumatic diseases). As well as complete medical examinations and sigmoidoscopic examination of the colon, a great many different laboratory tests and x-rays were carried out on Mr. Casina. The x-rays "showed none of the characteristic changes of fluoride, but again there was evidence of degenerative changes." Dr. Wightman continued: "the lumbosacral spine, for example, showed that there were five vertebrae with some minor disc degenerative changes in the lower lumbar spine. There is also evidence of unilateral articulation of the left transverse process with the sacrum . . ." "The left transverse process is a congenital abnormality which is not really of any significance." "The disc de-

generation again is a question of age and repeated trauma and possibly some metabolic change." Further evidence presented by Professor Wightman showed: "x-rays of his bowel and they were normal"; "we x-rayed his ankles and they were normal and x-rayed his chest and it was normal." Dr. McCarthy (specialist in rheumatic diseases) reported on his examinations to Dr. Wightman and wrote, in part, "Mr. Casina's symptoms on examination suggest the possibility of spondylitis [a type of arthritis of the spine], with peripheral joint involvement such as may be seen with psoriasis, colitis or possibly an infectious disease such as brucellosis; however, there is no radiological evidence of this condition. The present elevated serum uric acid, taken in conjunction with the initial symptoms of ankle pain and swelling, might suggest an atypical acute gouty attack . . ." "Repeated estimations of serum uric acid and uric acid in the urine were persistently elevated." As mentioned above, the x-rays of his bones were normal, his serum calcium and phosphorus were normal, "his alkaline phosphate enzyme having to do with bone, was normal." His serum electrolytes were normal. His "kidney function was normal." Thyroid function tests were normal and his urinary fluoride, on three different 24-hour samples, gave values of 0.4, 0.2, and 0.3 mg. F/l. The tentative diagnosis of Mr. Casina's conditions was that "we felt that he might have a gouty diathesis and it might be wise to treat him on the basis on that diagnosis."

264. On the basis of the evidence presented by Drs. Greene, Marson and Professor Wightman, and the examinations conducted by recognized clinical, radiological and laboratory specialists, there was no evidence which would suggest that Mr. Joseph Casina, Sr., had or has fluorosis—regardless of the fact, that, apparently, he does not wish to believe it.

MR. JACOB VANDERBEEK

265. Mr. Vanderbeek, farmer, resides 2½ miles NE of Port Maitland, R.R. #6, Dunnville (evidence was given through his son-in-law who acted as interpreter).

266. Summary of previous medical history: Dr. Rigg gave evidence to the effect that he had seen Mr. Vanderbeek in March, 1965, at which time he was not in good health. Dr. Rigg did not see the patient again until October, 1965, at which time Mr. Vanderbeek suggested that his complaints might be due to fluoride. He referred the patient to Dr. Marson in Hamilton, where extensive tests were carried out. He was seen again by Dr. Rigg in March, 1966, at which time Dr. Rigg noted that Mr. Vanderbeek had:

"general fatigue, felt dizzy, had pain in his chest, dreaming a lot at night." Dr. Rigg stated, "he is not a very healthy man," nor was he the first time Dr. Rigg saw him. He was seen again by Dr. Rigg in June, 1967, at which time "he had phlebitis."

267. Dr. Marson, to whom Mr. Vanderbeek was referred, admitted him to Chedoke Hospital on October 1st, 1965. The clinical history was obtained at that time through the services of a Dutch nurse, who acted as interpreter. Dr. Marson recalled that "the bed was literally covered with pamphlets, cuttings and various articles, all of which, as far as I could see, came from the States . . . the subject of fluorosis." From his notes, Dr. Marson read: "this patient who suspected that he himself is suffering from fluoride exposure (he did have some lame cattle) states that his health was good until the spring of '65 except that he tired readily for two years. It appears that he had occasional lower chest pain . . . lasting a few minutes but apparently unrelated to exertion and recurring three or four times per week. I believe he was complaining of this pain earlier in the year and that Dr. Rigg felt he might have heart trouble and had put him on digitalis and diuretics." "He states that he has swelling on the right wrist for the past week." "He then went on to state that for three years he has had pain in both thighs going towards the knee, and this does not shoot down the legs, is worse when he is working than resting and comes and goes. He has occasional difficulty in walking on this account. It is not affected by coughing and occurs most days and is unrelated to weather." On examination, the patient "had the physical signs, compatible with the diagnosis of chronic bronchitis. There was quite marked swelling of the dorsum of the right hand and lower forearm and pitting oedema over the hand and his wrist appears somewhat painful on passive movement. His back moved well and so did his hips."

268. Dr. Marson, continuing to read from his notes, said: "since the patient was disturbed about the possibility of fluoride exposure himself, naturally we went very carefully in the matter of investigations." X-rays were taken of the chest, spine, pelvis, both knees, etc. These x-rays "showed gross changes of osteoarthritis in the spine and left hip, gross disc disease, but there were no radiological findings to suggest fluorosis." Dr. Marson indicated that the Radiologist of Chedoke Hospital "went to great pains to look up the radiological aspects of this condition, reviewed the x-rays time and again, and there was no radiological evidence to suggest fluorosis." A 24-hour urine sample was sent to the Department of Health Laboratory in Toronto "for its fluorine content." "The

answer was 0.3 milligrams per litre." In reporting to Dr. Rigg, Dr. Marson wrote, in part, "I think we therefore assume that this patient has not been exposed to excessive fluorides."

269. Mr. Vanderbeek, according to Dr. Marson, had chronic bronchitis and gross osteoarthritis. He had, too, quite understandably, an anxiety brought about by the knowledge that some of his cattle had developed lameness as a result of excess fluoride. His own lameness could, Mr. Vanderbeek felt, be a comparable condition.

270. Mr. Vanderbeek was referred subsequently by his family physician to Professor Wightman and was admitted, by arrangement, to the Toronto General Hospital. X-rays were taken of the chest, pelvis, spine, femurs, tibias and fibulas, etc. The x-rays showed that "there is a minor degree of lipping of the articular margins of the hip and knee joints" and "degenerative changes in the lumbar spine and the thoracic spine." Dr. Wolfe (specialist in chronic chest conditions) carried out pulmonary function tests which "indicated that there was some obstruction to the flow of air through the lungs . . . but that there was not very severe damage to the ability of the lungs to transfer oxygen and carbon dioxide back and forth from the blood." Dr. Wightman said, of this condition in his lungs, "it is a fairly common condition with people in later life and it may be due to a variety of things—repeated lung infections or probably inherited defects"; in relation to the possibility of Mr. Vanderbeek having symptoms which might be related to fluorosis, Dr. Wightman pointed out "there was certainly no evidence in the x-rays of the kind of change that is seen in those bones when fluoride intoxication over a long period has been present." Repeated 24-hour urine samples, taken under controlled hospital conditions, showed fluoride concentrations of 0.3, 0.2, and 0.3 mg. F per litre (approximately 0.3, 0.2, and 0.3 p.p.m. F)—the same results as obtained in October, 1965. Professor Wightman felt of Mr. Vanderbeek, "first, that he had chronic bronchitis and emphysema and, secondly, that he had osteoarthritis of mild degree involving his spine; thirdly, that he had evidence of a former phlebitis in one leg."

271. The evidence of Drs. Rigg, Marson and Professor Wightman, and the reports of intensive investigations conducted in hospital, indicated that Mr. Vanderbeek did not have fluorosis.

MRS. ETHEL PARKE

272. Mrs. Parke resides 1½ miles NE of Port Maitland. She was referred by Dr. Mills to Professor Wightman and, being in

the Toronto General Hospital at the time of the hearings, was not available to give evidence. Professor Wightman, however, reported in full the investigations carried out on Mrs. Parke at the hospital. The results of those studies were submitted to the Committee. This summary is compiled from those reports.

273. Summary of previous medical history: Mrs. Parke, 57 years of age, feeling that she may be suffering from exposure to excessive fluoride, "was admitted to hospital for the investigation of her complaints and to determine whether she was suffering from acute or chronic fluoride poisoning." Mrs. Parke had had a uterine suspension done in 1937. In 1945, she "developed menorrhagia which was treated with radiation therapy." A severe reaction confined her to bed for six months. In 1945, she also had meningitis and, in 1958, "a diagnosis of hyperthyroidism was made and she was treated with radioactive iodine. She began to be hyperthyroid six months later and has been on thyroid since . . ." "In 1964, she again became very nervous with anorexia and insomnia . . . and had a feeling of pressure on her chest when she swallowed. She was evidently treated with tranquilizers then which seemed to help to some degree." "In September, 1965, she developed severe anterior chest pain radiating into her back and down her left arm. It was felt that she had a cardiac infarct and was kept in hospital for six weeks, at which time this same pain recurred so that a further six weeks was required in hospital." In December, 1966, "she developed, rather rapidly, a progressive arthralgia and eventually she had pain in her neck, the base of her skull, elbows, hands, knees and fingers." Other complaints were also noted.

274. In the summer of 1967, she had been referred by Dr. Mills to Dr. Marson, who admitted her to the Chedoke Hospital in Hamilton "for treatment of arthritis with physiotherapy." "In October, 1967, she developed Dupuytren's contracture, first in her right hand and then in her left." Not infrequently she has found "her temperature elevated to 99° or 101°." The pain in her right ear is "probably due to external otitis." "She has recurring sore throat." She "tends to be thirsty with polyuria." "She is allergic to penicillin, sulphonamide, horse serum and aspirin." Living about 1¼ miles NE of Port Maitland, she is "aware of irritating fumes in the air which seems to come from the smoke stack, but the greatest problem seems to be with the dust which blows over from the lagoon."

275. In the Toronto General Hospital, when Mrs. Parke was taken off thyroid medication, "she became obviously myxedematous

in a very short time." Numerous investigations were carried out. "The results may best be summarized by saying that she had a persistent mild anemia with an elevated sedimentation rate. She was obviously hypothyroid . . . her electrocardiogram showed . . . non-specific changes suggestive of some sort of diffuse myocardial disease."

276. Fluoride determination of 24-hour samples of urine showed concentrations of 1.0 mg F/l and on the next day 0.4 mg F/l, which are quite normal. "X-rays of the spine, pelvis and forearms did not show any evidence of calcification of ligaments or new bone formation or any other evidence of fluorosis from a radiological point of view." "On the other hand, while we felt convinced that she was not suffering from fluoride poisoning, we were unable to arrive [at] a diagnosis which explained all her symptoms. It seemed obvious that she had a reactive disorder of some sort, resembling rheumatoid arthritis."

277. A most intensive investigation was carried out on Mrs. Parke. She was examined not only by Professor Wightman, but as well by Dr. McCarthy, "who felt that she did not suffer from rheumatoid arthritis . . ."; by Dr. Yendt who found "no evidence of pelvic pathology." This evidence, together with the evidence of Drs. Mills and Marson, indicates that Mrs. Parke is suffering from something more than one clinical condition, but the evidence conclusively shows that she is not suffering as a result of excessive fluoride exposure. Mrs. Parke does not have fluorosis.

MR. TED BOORSMA

278. Mr. Boorsma, age 35, a farmer, resides 3¼ miles NE of Port Maitland.

279. Summary of previous medical history: Dr. Greene, in his evidence, stated that he had seen Mr. Boorsma "off and on for a number of years" and "that [in 1963] this boy had not been well." "He has had high blood pressure for years" and, on September 12th, 1967, he was "feeling weak and going downhill." Dr. Greene had noted signs and symptoms of hypertension, in 1963, and when he saw Mr. Boorsma in 1967 he found that he had anemia and recognized that the young man was quite ill. He investigated the anemia by doing blood smears and also bone marrow studies. However, "the reports from the pathology laboratory indicated a normal bone marrow."

280. Mr. Boorsma, on the basis of the arrangements made by the Department of Health and referred by Dr. Greene, was admitted to the Toronto General Hospital in mid-November, 1967, under the care of Professor Wightman. Professor Wightman explained that, since Mr. Boorsma was found to have an elevated serum calcium, a therapeutic trial, using prednisone, a cortisone-like drug, was initiated "because we thought he might have what is called sarcoidosis. We have not been able to confirm that diagnosis and he has not responded to the prednisone. The serum calcium and the urine calcium are persistently elevated and it looks as if he might have a disease called hyperparathyroidism due to overcapacity of a gland in the neck." (The parathyroid is a small gland lying close to the thyroid gland, which controls the calcium metabolism in the body.)

281. Subsequently, on April 1st, 1968, Professor Wightman reported the results of the detailed studies on Mr. Boorsma, conducted in the Toronto General Hospital over a period of more than three months. There it was found that urinary fluorides, on 24-hour samples, were consistently within the normal range, being on three consecutive periods 0.4, 0.2, and 0.5 mg/litre. "X-rays of chest, lumbar spine, shoulders, cervical spine, knees and ankles and forearms were all normal." "A liver biopsy was carried out to see if he had sarcoid and it was normal too." The "electrocardiogram showed some changes suggestive of left ventricular hypertrophy and possible posterior ischemia." "Plasma serum studies have been carried out to see if they would throw any light on the mechanism of his hypertension." Dr. D. McCarthy, Consultant to the Toronto General Hospital, also saw Mr. Boorsma and stated, in part, "his hypertension appears fairly prominent in a young man." Dr. Wightman concluded his report with the statement: "So far the only diagnosis we can make is hypercalcemia due to unknown cause, probably parathyroid adenoma. There is no evidence of fluoride intoxication." On the basis of the evidence presented, supported by the results of exhaustive tests, it can be stated that Mr. T. Boorsma did not have fluorosis.

282. No other residents of the area came forward voluntarily or were subpoenaed to give testimony relative to human health hazards. The summary of the evidence presented by three examining physicians, Dr. Greene, Dr. Mills and Dr. Rigg, the evidence presented by the Consultant, Dr. Marson, the evidence presented by Professor Wightman, and supported by x-ray examination of the patients and by urinary fluoride studies shows, as conclusively as it is clinically possible, that none of the several persons exam-

ined has been exposed to excessive concentrations of fluoride and none has fluorosis.

283. These findings are supported by a vast scientific and medical literature. The Committee hopes that the results of these intensive examinations will prove to the people of the Port Maitland area that, in spite of the statement of Dr. Waldbott (as quoted in the *Hamilton Spectator*, of Saturday, October 21st, 1967) that "two of nine farmers living within three miles of the plant are dying from fluorine poisoning," and in spite of Mr. Gosnell's statement (p. 31 of the script of the television film "Air of Death," October 22nd, 1967) "what's going to happen to these people who have fluoride poisoning," no one in the area has been exposed to fluoride, either through the air, water or food, in sufficient quantities to produce any clinical signs or symptoms suggestive of "fluoride poisoning" or of "chronic fluorosis" or of "crippling fluorosis." In other words, there is no human health hazard in the Port Maitland area directly or indirectly attributable to emissions from the ERCO plant and/or the Sherbrooke Metallurgical Company plant.

284. It is felt that the significance of these findings warrants the inclusion in this report of pertinent direct evidence given by the several physicians in connection with Mrs. Parke, Mr. Vanderbeek, Mr. Casina, Sr., and Mr. Boorsma. The complete and lengthy evidence is to be found in transcribed form in Volumes I, II, and III, pages 100 through 501, of the hearings.

DR. C. B. MILLS

285. Dr. C. B. Mills is a family physician, with offices in Dunnville. He is also the Medical Officer of Health for Sherbrooke and Dunn Townships. The following are pertinent excerpts from the evidence which he gave at the hearings:

Mr. Gordon: (p. 106)	*Do you feel competent to make any comments on the symptoms of fluorosis?*
Dr. Mills:	*I do not.*
Mr. Gordon:	*Have you at any time made any comments on the symptoms of fluorosis to any of your patients?*
Dr. Mills:	*I have commented that, reading some articles, I did not feel that the water samples,* per se, *indicated that they would be ingesting enough fluoride to develop fluorosis.*

Mr. Gordon:	*As a Medical Officer of Health, would you report your findings on matters relating to possible fluorosis that might be brought to your attention, or would these be reported to the Department of Health in Toronto?*
Dr. Mills:	*The only reports written were reports regarding the complaints and the question was then directed to the Department as to whether they felt that contamination did exist. Other than that, when Dr. Tidey came recently to investigate I told him of the complaints, that people had suggested to me, mostly indirectly, chiefly of stiffness of the joints.*
Mr. Gordon:	*Did these individuals personally come to you, or was this information that was relayed to you by another person?*
Dr. Mills:	*There were a few individuals that I saw in this regard.*
Mr. Gordon:	*Could you give us their names?*
Dr. Mills:	*Mr. and Mrs. Don MacKeigan . . .*
Mr. Gordon:	*Is that all?*
Dr. Mills:	*No, there are others. As I mentioned, Mr. William Siddall, and I examined his wife also, and his daughter-in-law, Mrs. Wayne Siddall. There was one other couple, but I can't recall the name at the moment.*
Mr. Gordon: (p. 108)	*Did you receive a visit from Mr. Tidey in or about the month of October, 1965?*
Dr. Mills:	*Yes.*
Mr. Gordon:	*Did you also discuss with Dr. Tidey at that time the question that you were concerned about milk from cows that didn't like the grazing in Moulton or Sherbrooke Townships?*
Dr. Mills:	*The subject was discussed to a lesser degree, because I was unable to have figures on it.*

Mr. Gordon:	*And did Dr. Tidey indicate to you in October of 1965, based on his assessment, there was or there was not any danger to milk that would be produced from cattle grazing in the area?*
Dr. Mills:	*I do not believe that he had those figures at that time to make that statement.*
Mr. Gordon:	*Were you aware of any health problems involving local residents in October of 1965?*
Dr. Mills:	*No.*
Mr. Gordon: *(p. 109)*	*And did you so advise Dr. Tidey?*
Dr. Mills:	*Yes.*
Mr. Gordon:	*In the month of October, 1965, did you arrange with Dr. Tidey that if any health problems did arise you would notify his Department?*
Dr. Mills:	*Yes.*
Mr. Gordon:	*Did any health problems arise after that meeting?*
Dr. Mills:	*None, until the question arose again when Dr. Tidey came here this past fall.*
Mr. Gordon:	*So that, as Medical Officer of Health, from the month of October, 1965, up to the fall of 1967, you personally were not aware of any health problem involving local residents from pollutants?*
Dr. Mills:	*None, with the possible exception of the application of Mr. Zynomirski stating that the cabbages which he had prepared for use during the winter were not fit to eat; whether he felt it was because of its appearance or because of some direct effect on his own digestion, I do not recall, but this would be the only possible exception.*
Mr. Gordon:	*And that was a problem involving the effect of any possible pollutant on vegetation?*

Dr. Mills:	*Yes.*
Mr. Gordon:	*Not on human health?*
Dr. Mills:	*No.*
Mr. Gordon:	*Since last fall and up to the present time, have you had any patients who have come to you complaining specifically of suffering from fluorosis?*
Dr. Mills:	*No.*
Mr. Gordon:	*Are you aware of any people in the area—in your capacity as Medical Officer of Health—of anyone suffering from fluorosis?*
Dr. Mills:	*I am not aware of the diagnosis of fluorosis having been made on any person.*
Mr. Gordon:	*Much had been written in the press concerning this problem and also on the radio and TV. Were you interviewed at any time by the press concerning a fluorosis problem in the Dunnville area?*
Dr. Mills:	*I gave them figures, I gave the press figures which had been submitted to me, as Medical Officer of Health, on several water samples, which reports I have copies of. The names I did not give, but rather general figures to show that the particular samples in question had a low fluoride content.*
Mr. Gordon: (p. 113)	*Then, in summary up to the month of October, 1967, as the qualified Medical Officer of Health for Sherbrooke and Dunn, you were not aware of any problem concerning human beings affected by any pollutants?*
Dr. Mills:	*I was aware of complaints that supposedly . . . that people stated could be due to it, but I was not aware of the specific diagnosis of fluoride.*
Mr. Gordon:	*And you had not seen any patient or individual who was specifically diagnosed as having fluorosis?*
Dr. Mills:	*No.*

Mr. Brooks: (p. 116) — *Did any of these people . . . I refer specifically to Mr. Casina, Mrs. Ethel Parke . . . are they any of your patients?*

Dr. Mills: — *Mrs. Ethel Parke.*

Mr. Brooks: — *Did she come, I am referring to your records, did she approach you at any time between 1960 and 1965 regarding disabilities which subsequently might be related to symptoms of fluoride poisoning?*

Dr. Mills: (p. 118) — *If I may comment, Mr. Brooks, at this point I referred, as is known, Mrs. Parke to Toronto some months or so ago* [27th January, 1968]. *I have no knowledge of what has transpired since that time. I could not, therefore, refer any symptoms, as Dr. Hall has mentioned. I stated that I did not know of any symptoms in any patient that had been diagnosed as fluoride and consequently, not knowing fluorosis symptoms more specifically, that statement would stand in this regard, to my knowledge of Mrs. Parke.*

Mr. Pepper: (p. 123) — *Dr. Mills, I gather you are not only a Medical Officer of Health, but that you also practise as a general practitioner?*

Dr. Mills: — *Yes I am, a general practitioner.*

Mr. Pepper: — *So I take it that the effect of your evidence is that neither in your capacity as Medical Officer of Health nor as a general practitioner in the area do you know of any evidence of any person suffering from the result of fluorides?*

Dr. Mills: — *I do not.*

DR. F. G. RIGG

286. Dr. F. G. Rigg is a family physician with offices in Dunnville. The following excerpts from his evidence are pertinent to his association with the two patients, Mr. Vanderbeek and Mrs. Nie.

Mr. Gordon: (p. 126) — *During the term that you have practised in Dunnville, have you had any patients who*

	have come to you and complained of any effects of fluoride?
Dr. Rigg:	*I have had patients who said that they had trouble from fluoride.*
Mr. Gordon:	*Do you recall how many patients thought they had trouble?*
Dr. Rigg:	*Two.*
Mr. Gordon:	*And how long ago was that?*
Dr. Rigg:	*One was about two and a half years ago, and one was about six months ago.*
Mr. Gordon:	*And could you give us the name of that person?*
Dr. Rigg:	*Mr. Vanderbeek.*
Mr. Gordon:	*He consulted you about two and a half years ago thinking that fluorides had some effect upon his health?*
Dr. Rigg:	*That is right.*
Mr. Gordon:	*Upon hearing this, what did you then do?*
Dr. Rigg:	*I referred him to Hamilton for investigation.*
Mr. Gordon:	*To what person in Hamilton?*
Dr. Rigg:	*Dr. Marson.*
Mr. Gordon:	*After his release from hospital in Hamilton, when did he again come to see you?*
Dr. Rigg:	*March, 1966.*
Mr. Gordon:	*And did he have a complaint at that particular time?*
Dr. Rigg:	*Yes.*
Mr. Gordon:	*What was the nature of that complaint?*
Dr. Rigg:	*Just general fatigue, felt dizzy, had pain in his chest, dreaming a lot at night.*
Mr. Gordon:	*What was his general physical health?*
Dr. Rigg:	*He is not a very healthy man.*

Mr. Gordon:	*Was he a healthy man when you first saw him.*
Dr. Rigg:	*No, he wasn't.*
Mr. Gordon: (p. 129)	*So after the consultation, did he come to see you again?*
Dr. Rigg:	*Yes.*
Mr. Gordon:	*And for what purpose?*
Dr. Rigg:	*He had phlebitis at that time.*
Mr. Gordon:	*Did you have any other patients who came to you specifically claiming they might have had some ill effect from fluorides or claiming that they had fluorosis?*
Dr. Rigg:	*Nobody that came complained that they had fluorosis. One patient thought she might have some difficulty as a result of fluoride when she was in for other complaints at the office.*
Mr. Gordon:	*And what did you recommend for that particular patient?*
Dr. Rigg:	*I suggested she go to Toronto for investigation.*
Mr. Gordon:	*What was the name of that patient?*
Dr. Rigg:	*Mrs. Shirley Nie.*
Mr. Gordon:	*Other than Mr. Vanderbeek and Mrs. Shirley Nie, did you have any other patients complaining of the effect of fluorides?*
Dr. Rigg:	*No.*
Mr. Gordon: (p. 130)	*So that prior to October, 1967, other than the two patients whose names you have given to us, you didn't really have anyone come in to you complaining about having fluorosis?*
Dr. Rigg:	*That is right.*

DR. C. B. GREENE

287. Dr. C. B. Greene is a family physician with offices in Dunnville. His evidence at the hearings dealt primarily with two of his

patients, Mr. J. Casina, Sr., and Mr. T. Boorsma. Particular excerpts from the evidence are presented here.

Mr. Gordon: *(p. 144)*	*This inquiry is concerned with allegations that have been made concerning possible fluoride poisoning resulting in a disease known as fluorosis. Have you had any patients who have come to you while you have practised in Dunnville complaining of the effects of fluoride upon them?*
Dr. Greene:	*I have.*
Mr. Gordon:	*How many?*
Dr. Greene:	*I have three who specifically said they had fluoride involvement. There were others who came for general examinations because they were advised to because they lived in the area, not because they had any particular symptoms.*
Mr. Gordon:	*They resided in what area?*
Dr. Greene:	*In the area of the Port Maitland plant.*
Mr. Gordon:	*Did they come to you after October, 1967?*
Dr. Greene:	*The ones who came in for general checkups, yes.*
Mr. Gordon: *(p. 145)*	*And prior to the month of October, 1967, how many persons had come to you specifically with a fluoride problem?*
Dr. Greene:	*Two.*
Mr. Gordon:	*And who were they?*
Dr. Greene:	*Mr. Joseph Casina and Mr. Ted Boorsma.*
Mr. Gordon:	*And at that time* [July 7th, 1967] *did Mr. Casina tell you that he thought he had fluorosis, or did he ask you to make a diagnosis to determine whether he had?*
Dr. Greene:	*He felt very strongly at that time that it was a good possibility that he had reason to believe that he lived in this contaminated area and that he might possibly be suffering from the effects of it.*

Mr. Gordon:	*As a result of what he told you, what did you do?*
Dr. Greene:	*We examined the man, I examined him and he was complaining at that time of pains in his ankles with some swelling in his right ankle and, having taken this into consideration, I consulted with the Department of Health what my steps should then be.*
Mr. Gordon:	*When was this?*
Dr. Greene:	*This was in July, 1967. Blood samples and urine samples were taken and forwarded to the Department of Health.*
Mr. Gordon: (p. 146)	*How long had he* [Mr. Casina] *been a patient of yours?*
Dr. Greene:	*Since 1962, October 3rd, 1962.*
Mr. Gordon:	*What was his general health when he first came to see you?*
Dr. Greene:	*In 1962, Mr. Casina was a fairly robust farmer. He was a big man and was in good health up until 1964.*
Mr. Gordon:	*Do you know what happened in 1964?*
Dr. Greene:	*At that time he developed a coronary.*
Mr. Gordon: (p. 147)	*After he came to you and indicated that he thought he might be suffering from fluorosis you sent the samples of urine and blood to be examined, is that correct?*
Dr. Greene:	*That is correct.*
Mr. Gordon:	*Did you yourself do anything further in that connection?*
Dr. Greene:	*Yes, I sent him to a consultant, Dr. F. Graham Marson of Hamilton.*
Mr. Gordon:	*And did you work in connection with the consultant?*
Dr. Greene:	*He sent me his report on examination of the man that he could find, so far as he was concerned, no evidence to suggest that this man had fluorosis.*

Mr. Gordon:	*And then did you relay that information to Mr. Casina?*
Dr. Greene:	*This was done.*
Mr. Gordon:	*Did he come back to you after that information had been relayed to him?*
Dr. Greene:	*Yes, this man came back because he had some swelling of the ankles and very painful ankles and knee, so much that he was going about with a cane. The matter was investigated further, x-rays were taken of his legs, his knee joints, his ankles and so on and so forth, and the x-ray reports that I have available by the radiologist he could find no definite evidence of bone pathology.*
The Chairman: (p. 148)	*Excuse me, may I ask Dr. Greene a question? Was his myocardial function improving through 1967?*
Dr. Greene:	*That is right. At times he became very apprehensive. Naturally, he was very worried over this condition, this fluorosis, and this sensationalism that was going on at the time and he was very concerned about his health and, of course, a man who has had a previous coronary this concerned me much more than anything else, because this man was beginning to have symptoms which might lead to a second episode which might not have been so pleasant.*
Mr. Gordon: (p. 149)	*Upon learning this fact, did you feel there was any treatment, any form of pills that could be given to alleviate this condition?*
Dr. Greene:	*Yes, we then thought in terms of a possible gouty condition and although he did have an elevated uric acid—this is the chemical usually responsible for gout—was high, there was not yet any physical thing actually to demonstrate that he did have clinical gout . . .*
Mr. Gordon:	*When did he* [Mr. T. Boorsma] *first consult with you?*

Dr. Greene:	*I had seen Mr. Boorsma off and on for quite a number of years. This boy had not been well. He suffered from high blood pressure for years and the first time this boy came in really complaining of feeling weak and going downhill, the first recorded time I have here was September the 12th, 1967. He was complaining of general lassitude, tired, not able to do as much work as he usually did . . . he had more or less vague aches and pains all over, and just a general debility and obviously that man looked sick.*
Mr. Gordon:	*Had he been a patient of yours prior to this time?*
Dr. Greene:	*He had been attending my office since my coming to Dunnville, and the first recorded visit I have had from this gentleman was back in the year 1963, November 15th, 1963.*
Mr. Gordon:	*What was his physical condition at the time?*
Dr. Greene:	*Generally speaking, this man has had considerable hypertension for a man of his age. He had some evidence of eye abnormalities insofar as he used to complain of his vision being fuzzy, and there was some calcification in his blood vessels, which is rather unusual unless there is something amiss in a man that young . . . 26 years of age.*
Mr. Gordon: *(p. 151)*	*Did he* [Mr. Boorsma] *go to Toronto on your advice or on the advice of somebody else?*
Dr. Greene:	*It was an arrangement made between myself—because when these two patients came in I immediately got in touch with the Department of Health and worked under their supervision insofar as to what I should do in carrying on the investigations. In the case of Mr. Boorsma, I went a little further because I thought this man was sick. He had some degree of anemia, which was unusual, and his was investigated from blood smears and also from bone marrow.*

Mr. Gordon:	*Where did you send these smears?*
Dr. Greene:	*The St. Catharines General Hospital who do the pathology for our hospital.*
Mr. Gordon:	*Other than those two patients that you have just described* [Mr. Casina and Mr. Boorsma], *prior to October, 1967, had anyone else come to you complaining about the effects of fluoride?*
Dr. Greene:	*I saw one man whose name had already been mentioned here this afternoon, a Mr. Vanderbeek.*
Mr. Gordon:	*How long ago was that?*
Dr. Greene:	*That was on October 27th, 1967.*
Mr. Gordon:	*Had he been a patient of yours prior to that time?*
Dr. Greene:	*No.*
Mr. Gordon:	*And as a result of that visit to you on October 27th, what did you recommend that he do?*
Dr. Greene:	*I recommended that he have the same investigation as the other two previous men. I examined him generally, physically, and my findings are much in agreement with the previous witness, as far as his general health is concerned. I advised him to have the full investigation under the direction of the Department of Health, which he subsequently did. He went with the other two men.*

DR. F. G. MARSON

288. Dr. F. Graham Marson is a member of the active staff of the Hamilton Civic Hospital, Chedoke Hospital in Hamilton, and is associated with the Dunnville Hospital, the Grimsby Hospital and the Brant and St. Joseph's Hospital as a Consultant in the field of general internal medicine, with special experience and interest in rheumatology and gastro-enterology, and has acted as a Consultant to the Doctors in the Dunnville area since 1958.

289. The following evidence was given by Dr. Marson at the hearings of Mr. Jacob Vanderbeek and Mr. Joseph Casina, Sr.:

Mr. Gordon: *(p. 364)*	*Recently were you contacted by any of these Doctors and did they refer any of their patients to you?*
Dr. Marson:	*Yes, I have seen two patients from the Dunnville area referred on the basis of possible fluoride exposure.*
Mr. Gordon:	*How long ago was that?*
Dr. Marson:	*The first one—you will have to excuse me reading from the records, but I have not seen this person since this time and this was back in October, 1965.*
Mr. Gordon:	*Who was the person that you saw?*
Dr. Marson:	*The person was Mr. Jacob Vanderbeek.*
Mr. Gordon:	*Prior to that date, had you been consulted by any of the Doctors from the Dunnville area concerning the possibility of fluoride poisoning in humans in this area?*
Dr. Marson:	*No, this was the first mention I ever heard of fluoride exposure or possible exposure to man in this area, the first mention.*
Mr. Gordon:	*And who referred him to you?*
Dr. Marson:	*Dr. Frederick Rigg.*
Mr. Gordon:	*Did Mr. Vanderbeek attend at your office?*
Dr. Marson:	*No, we took him straight into the Chedoke General Hospital and I studied him whilst he was in there; he was under my care as coming from outside the town.*
Mr. Gordon:	*Just tell us what occurred while he was in the hospital, what you did by way of treatment?*
Dr. Marson:	*The first thing I did was try and extract a good history and this was immediately a problem because we required the use of an interpreter but, with the use of the services of a Dutch nurse as interpreter, we did obtain quite a history. It was a very dramatic meeting, I might say, I will never forget it.*

	I remember the patient's bed very well and I remember once seeing the bed—covered in pamphlets on this subject.
Mr. Gordon:	*On what subject?*
Dr. Marson:	*The subject of fluorosis. The bed literally was covered with pamphlets, cuttings and various articles, all of which as far as I could see, came from the States. I could quote some of these. But anyhow would you like the history?*

290. Dr. Marson presented the general history which he had taken of this patient, discussed the investigations which had been carried out on him, including 24-hour urine samples for fluoride analysis, x-rays of the chest, spine, pelvis, knees, etc., and the findings. He then continued:

Dr. Marson:	*These x-rays—and I discussed these with the Chief of Radiology at the time—showed gross changes of osteoarthritis in the spine and in the left hip, gross disc disease, but there was no radiological findings to suggest fluorosis. And I might say that I spent some time with the radiologist, who is a very respected gentleman, and we went to great pains to look up the radiological aspects of this condition, reviewed the x-rays time and again, and there was no radiological evidence to suggest fluorosis . . . The urine fluoride content came back to us in the course of time ... and the answer was 0.3 mg per litre (about 0.3 p.p.m.). My last sentence on the third and final letter to Dr. Rigg about this patient: "I think we, therefore, assume that this patient has not been exposed to excessive fluoride."*
Mr. Gordon: *(p. 371)*	*Were any other patients from the Dunnville area referred to you?*
Dr. Marson:	*The only other one is a Mr. Joseph Casina.*
Mr. Gordon:	*When was he first referred to you and by whom?*

Dr. Marson: *I have seen this gentleman twice professionally. The first time was on July 18th, 1967. The second time was on October 17th, 1967. I saw this gentleman on July 18th, 1967, in consultation at the request of Dr. A. H. Marshall. I very well recall this meeting because this was one of the most anxious people I have seen in a long time. I am not a psychiatrist, so perhaps they don't come my way, but he was extremely anxious and apprehensive, or so it seemed.*

291. Up to this time, it had been indicated in the evidence that Mr. Casina had had a coronary attack in 1964. However, when he was questioned later, it was found that he had had a previous heart attack in 1952.

Mr. Gordon: *How has your health been in the last 15 years?*

Mr. Casina: *That would be going back to 1952 or 1953. In 1952 I had a heart attack.*

Mr. Gordon: *(p. 462)* *Did you only have one heart attack?*

Mr. Casina: *Two.*

Mr. Gordon: *When was the second one?*

Mr. Casina: *1964.*

Mr. Gordon: *What is your present age?*

Mr. Casina: *55.*

Mr. Gordon: *After you had had your second heart attack, did you still continue to do the work on the farm?*

Mr. Casina: *Yes.*

292. Returning to Dr. Marson; in evidence, he quoted from his notes about Mr. Casina:

Dr. Marson: *(p. 373)* *He suggested to me he was fine until a month ago, but he has some discomfort in the right side of his chest during the past month. Some discomfort in the trapezii* [muscles which are part of the back of the shoulders] *and the back of the neck and low back pain*

and painful ankles, for two and a half weeks. He was walking with a stick and professed to have extreme pain in his ankles and talked about them feeling hot and that they had been swollen so that he had to increase the size of his shoes and indeed he stated they were still swollen, which fact I could not confirm . . . He confessed that he has been "nerved up" (that was his own expression) for a few years about the local fluorine problem.

Dr. Marson: (p 374) — *He* [Mr. Casina] *had a previous history which he gave me of a heart attack in August, 1964, and he told me that there was nothing else of significance, that there was no family history of gout or rheumatism . . . The ankles were not swollen to me at all, but at first almost touching them seemed to cause exquisite pain, but later, provided I engaged him in conversation, I could move them fairly freely without too much trouble. There was certainly no local heat or redness. I well remember this. This patient was quite adamant that his ankles were swollen in front of my eyes. I don't profess to be an expert in rheumatology and I could not personally see any swelling whatsoever. This happened again on the second meeting.*

293. Dr. Marson, in continuing his evidence, stated:

Dr. Marson: (p. 375) — *I am reading from my consultation at that time—I find it difficult to think that this patient has any underlying disorder responsible for his various symptoms, but I think he should have a sed rate, and I cannot be adamant that he does not have some condition such as rheumatoid in his ankles or even gout. He may require such things as serum uric acid and rheumatoid serology.*

Dr. Marson: (p. 377) — *Seeing this patient the first time, and treating patients as human beings and not as isolated joints, it seemed to me this patient was in a state of extreme apprehension*

which, as far as I knew, was very genuine and understandable and I thought it was extremely important to decide once and for all whether he could possibly be suffering from fluorosis. I didn't think he likely was, but I thought we had to make sure. I appreciated the potential implications of the future. So, when I learned that the blood level [fluoride level] *was normal, and this had not returned when I first saw the patient, it was my opinion that this patient could not be suffering any symptoms of fluorosis, that he did not have fluorosis and these symptoms, whatever they were due to, had nothing to do with fluorine exposure. I thought, in that he was in this state of apprehension, it was terribly important for that patient to be reassured, but here again as a Consultant to the Doctor, I was not personally in a position to do this reassuring, because I can't ring up the patient and ask him to come and see me again. . . But I did see Mr. Casina again on October 17th, 1967. I don't think that at this time I was specifically asked to see him with fluorine in mind, as far as I know. In fact, my understanding was that this had been settled. I did not think the question arose any longer, and I thought I was now seeing him as any other patient who had symptoms and they wanted diagnosis and suggestions re management. I might say the patient's demeanour on this occasion was altogether different; he was no longer in a state of apprehension or so it seemed to me.*

295. Dr. Marson continued with a description of the signs and symptoms exhibited by the patient and of his examination. Reading from his consultation notes, Dr. Marson continued:

Dr. Marson: *(p. 380)*

This is my writing here: The ankles looked totally unremarkable to me and I was unable to accept that they were swollen, although he insisted that they were. Now, at the time, not having any previous records, I didn't appreciate that I was duplicating a

remark that I had made a few months before and here we were again seeing a patient who insisted in front of me that his ankles were swollen, but in my personal opinion they were not.

And I have listed various investigations there which do not include further fluorine studies because, as far as I was concerned, that was not the issue and had been decided already and I say that . . . I do not know of any form of arthritis that presents itself like this, with pain in the ankles which radiates up to the knees and which does not show increased stiffness after resting and is not worse on walking than on resting.

Mr. Gordon: (p. 381) — *In summary, then, so far as the ankles were concerned, is there any medical term that you could apply to describe that particular ailment?*

Dr. Marson: — *I think we see patients who complain of severe pain in joints and yet, when we come to examine the joints, we don't necessarily find any objective signs. I don't think that has to mean that there isn't anything wrong with that joint necessarily, but when you see a patient twice over a period of a few months and on both occasions he insists that his ankles are swollen and you are there examining him at the time and, as an expert rheumatologist, you can find no evidence whatsoever for the swelling, it makes me wonder about the credibility and I think this is the sort of thing you have to take into account when you are trying to assess a patient.*

Mr. Gordon: (p. 382) — *So in Mr. Casina's case and in the case of Mr. Vanderbeek you did not diagnose, or as a result of the investigation you conducted, you did not ascertain any fluoride poisoning or evidence of it?*

Dr. Marson: — *On the basis of what I ascertained myself and the investigations that were done later,*

and I found the results as far as I am concerned, I am as certain as I can be of anything, that there was no evidence whatsoever that any symptoms that either of these gentlemen had or have were due to fluoride exposure.

Mr. Gordon:	*Do you think that Mr. Casina was suffering from any forms of what could be called pollution damage?*
Dr. Marson:	*No, I do not.*

DR. K. J. R. WIGHTMAN

295. Professor K. J. R. Wightman, Chairman of the Sir John and Lady Eaton Department of Medicine, University of Toronto, had certain patients referred to him by doctors practising in the area and, through arrangements made by the Department of Health, such patients were admitted to the Toronto General Hospital for intensive examination.

296.

Mr. Gordon: (p. 401)	*I understand that certain patients have been sent to you from the Dunnville area?*
Dr. Wightman:	*That is correct.*
Mr. Gordon:	*Would you tell us how many patients have been referred to you and when?*
Dr. Wightman:	*Four patients were referred. I can't tell you exactly when . . . these patients were sent to the University of Toronto, the first two, and then another two, and two of the patients are still in hospital.*
Mr. Gordon:	*Which two are still in the hospital?*
Dr. Wightman:	*Mr. Boorsma and a Mrs. Parke.*
Mr. Gordon:	*Then the two other patients who were sent to the hospital?*
Dr. Wightman:	*The other two patients were Mr. Casina and Mr. Vanderbeek.*

297. After describing various signs and symptoms relative to Mr. Vanderbeek, Dr. Wightman was asked:

Mr. Gordon: (p. 404)	*Were you aware of the fact that, at the time you were examining these patients, that complaints had been made concerning possible fluoride poisoning?*
Dr. Wightman:	*Yes.*
Mr. Gordon:	*From the reading that you have done, did Mr. Vanderbeek appear to have any symptoms which might be related to fluorosis?*
Dr. Wightman:	*I didn't think so. I thought that the only symptoms which might conceivably be related were the symptoms he had of pains in his joints and bones. There was certainly no evidence in the x-rays of the kind of change that is seen in those bones when fluoride intoxication over a long period has been present. We tested his urine for fluoride and I was told that the level was normal and really there was nothing to suggest that his symptoms required any other explanation, except the ones which we found which I would say were, first, that he had chronic bronchitis and emphysema and, secondly, that he had osteoarthritis of mild degree involving his spine. Thirdly, that he had evidence of a former phlebitis in one leg.*
Mr. Gordon: (p. 406)	*Did you inform Mr. Vanderbeek of the results of your investigation?*
Dr. Wightman:	*Yes.*
Mr. Gordon:	*Doctor, could you tell us what is the percentage of people in middle age who have chronic bronchitis or emphysema?*
Dr. Wightman:	*Chronic bronchitis or emphysema ... well, in middle life and later, it seems to be increasing in our population. I can't really tell you the percentage but it might be 10 or 20. I don't know the exact answer. It is one of the diseases in an aging population which is causing the problem and we are thinking now that we will need greater facilities for taking care of people with the disease. So*

	it is a growing problem, but it certainly is a common disease.
Mr. Gordon: (p. 409)	*What information did you receive concerning his* [Mr. Casina's] *condition so that you could conduct your investigations?*
Dr. Wightman:	*Well, I was told the same thing, that there was a problem here and that he had been seen previously by other physicians and that it was his desire and that of his family doctor to have his condition further investigated.*
Mr. Gordon:	*Would you tell us what work you were involved with, with this particular patient during that period?*
Dr. Wightman:	*I conducted a sort of initial survey of his condition and tried to sort out what symptoms were due to what. I asked various people to see him with me, including Dr. Yendt and Dr. McCarthy, and again, with the idea of having these calcium fluoride studies done, also to see what the nature of his joint symptoms was I carried out a sigmoidscopic examination of his colon because he had symptoms of his bowel and I think that fairly well summarized what was done. A great many laboratory tests and x-rays were carried out, and we have these x-rays here. I also have x-rays which were taken previously at the Haldimand Hospital, which were sent to us for comparison with our x-rays.*
Mr. Gordon:	*Were any abnormalities noted in these x-rays?*
Dr. Wightman:	*Again, they showed none of the characteristic changes of fluoride, but again there was evidence of degenerative changes.*
Mr. Gordon: (p. 411)	*While you are giving us these, could you tell us what may have caused this condition?*
Dr. Wightman:	*Well, first of all, the thing about the left transverse process is a congenital abnormality which is not really of any significance. It*

	is just a curious way that his spine has developed.
	The disc degeneration, again, is a question of age and repeated trauma and possibly some metabolic change, that some individuals seem to be more susceptible to than others.
Mr. Gordon: (p. 418)	*You have pretty well summarized the medical evidence that you have concerning the two patients, is that correct?*
Dr. Wightman:	*I think that there is nothing else to say. If I may just look at the letter that I wrote to the Doctor—we suggested some therapy or treatment, suggested that he change his diet, that he have some antispasmodic; we felt that he might have a gouty diathesis and it might be wise to treat him on the basis of that diagnosis.*
Mr. Gordon: (p. 421)	*Then in summary the reports for Mr. Boorsma and Ethel Parke should be available in another three weeks to a month?*
Dr. Wightman:	*I should think so, yes.*

298. The report for Mrs. Ethel Parke was received by the Committee on March 29th and that for Mr. T. Boorsma on April 1st. Comments in connection with these two patients have already been given. The final diagnosis of each of these patients likewise is contained in the discussion relative to them; they are added here in a brief form so that they might be included with the diagnoses of the conditions complained about by Mr. Casina and Mr. Vanderbeek.

(a) In connection with Mrs. Parke's case, the Consultants of the Toronto General Hospital could find no evidence that she had been suffering from fluoride poisoning. The final diagnosis on her was: "Reactive disease NYD, anaemia; elevated sedimentation rate."

(b) In the case of Mr. Ted Boorsma, again the Consultants of the Toronto General Hospital found no evidence of fluoride intoxication. The diagnosis which they have made is: "Hyper-calcemia, due to unknown causes, probably parathyroid adenoma."

299. Special attention was naturally given to those residents of the area who felt that they might be "suffering from fluorosis." Urine samples obtained by local and consulting physicians, from these persons who were concerned about the possibility of having chronic fluorosis as a result of "alleged" pollution, were analyzed from time to time. On no occasion did any of the samples from these special persons show a higher than normal fluoride concentration, in either single spot samples or in 24-hour samples taken under hospitalized and controlled conditions. In connection with the four resident area persons who took advantage of the Department of Health's offer to be hospitalized in Toronto General Hospital for thorough examination by Professor K. J. R. Wightman, at no cost to themselves, controlled urine specimens were taken and analyzed for fluorides. The samples were "coded" before being sent to the laboratory. The patients' names were likewise "coded" and it was only after the hearings on the Human Health aspect of the Inquiry had been completed that the individual results of the urine analyses and the "code" were made known to the Commissioners. In each series of urine sampling, known quantities of fluoride were added to certain 24-hour samples in order to check the accuracy of the analysis. A summary of the results is presented in Table IX.

TABLE IX

SUMMARY—24-HOUR URINE SAMPLES

Decoded name of Patient	*Decoded Sample Number*	*Urinary F Mg/l*
Mr. Casina Sr.	1	0.4
Mr. Casina Sr.	5	0.2
Mr. Casina Sr.	12	0.3
Mr. Boorsma	3	0.4
Mr. Boorsma	7	0.2
Mr. Boorsma	10	0.5
Mrs. Parke	20	1.0
Mrs. Parke	21	0.4
Mr. Vanderbeek	13	0.3
Mr. Vanderbeek	16	0.2
Mr. Vanderbeek	17	0.3

300. These results, which are perfectly normal, are in keeping with earlier results obtained on urine samples submitted by their local physicians and the consulting physicians.

H. SUMMARY — EVIDENCE ON HUMAN FLUOROSIS

301. With regard to the intensive clinical and laboratory examinations conducted by the family physicians and by Clinical Consultants, and on the basis of the essential diagnostic criteria, none of the persons—Mrs. Parke, Mr. Vanderbeek, Mr. Boorsma and Mr. Casina, Sr.—had or has any clinical condition which was or is due to high fluoride ingestion. There was no evidence of fluorosis in these persons or in any of the other persons who appeared as witnesses at the hearings, thinking that they might be suffering from fluorosis, and who had been examined by any of the local physicians or consultants who gave evidence.

302. Samples of air taken in the "high risk" areas of the ERCO plant showed high concentrations of fluoride on several occasions. Urinary fluoride in a few of the workers in such areas was found to be in the high normal range and a few even in the above normal range. Internal plant control, in the use of masks by employees in such high risk areas, and improved control of conditions in the "bins" are strongly recommended. It is inconceivable that ERCO did not have a consulting physician to advise on the several aspects relating to the continuing good health of the employees.

303. It is of concern, from a total health point of view, that so many residents of the area use roof-run and cistern water for drinking purposes. The pathological bacteria count of several of the cisterns' water indicated a high bacterial contamination.

304. Some degree of upper respiratory irritation has been recognized in some people on several different occasions. It is suspected that these conditions coincided with the excessive emission of pollutants from either or both the ERCO Plant and the Sherbrooke Metallurgical Company Plant. In the latter case, considerably higher stacks would lessen the concentration of SO_2 in the area. In the former case, vastly improved control of the gypsum lagoon beds and of the emissions from the "bins" areas are essential.

I. CONCLUSIONS ON HUMAN HEALTH ON THE BASIS OF EVIDENCE AND LITERATURE

305. In order to diagnose adequately chronic fluorosis in humans, it is necessary that more than one of the following conditions be found to be present:

(a) a history of lengthy exposure to relatively high concentrations of fluoride in the air, food and/or water;

(b) severe mottling or staining of the permanent teeth;

(c) radiological evidence of exostosis of the bone;

(d) high fluoride concentration in the dry bone ash;

(e) above normal concentrations of fluoride in samples of urine taken over a period of time.

306. On the basis of these essential criteria the evidence, together with reports and briefs filed as Exhibits at the hearings, shows:

(a) (i) that concentrations of fluoride in the air of above acceptable levels were determined on several occasions but, recognizing that such samples contained quantities of particulate matter and that of such particulates only those of size less than 7 millimicrons can be absorbed into the lung, such occurences could not lead to human fluorosis;

(ii) that the fluoride concentration of domestic drinking water supplies was well below 1.0 p.p.m. From the data showing the fluoride concentration of water samples taken from 195 homes in the area, various school wells and municipal supplies, it is apparent that the residents of the area are not drinking water which is "high" in fluoride.

(iii) that the human consumption of meat, milk, vegetables and other foodstuffs grown in the area could not possibly produce fluorosis.

(b) that no witness mentioned "mottling of the teeth." However, the Committee asked that a dental survey of area children be conducted. This study carried out on 151 children in the area revealed that only one child had aesthetically significant mottling—this of "moderate" degree. There was no evidence of staining of the permanent teeth. Such an examination gives evidence that the maximum fluoride intake of these children during the period 1957 to 1964 was equivalent to that derived from drinking water containing 1.5 to 1.9 p.p.m. fluoride. This is the same or lower than many municipalities in Ontario.

(c) that there was no radiological evidence of exostosis of the bone of any of the several persons who felt that they might be suffering from fluorosis. These x-rays were carried out in the Dunnville Hospital, the Chedoke Hospital of Hamilton and in the Toronto General Hospital.

(d) that there was no reason to indulge in bone biopsy in order to determine the fluoride concentration in the dry bone ash —in view of a, b, c and e.

(e) that the fluoride concentrations of urine samples of those persons who felt that they had fluorosis were always normal; similar results were obtained when 24-hour urine samples were taken and tested under controlled hospital conditions.

307. It is comforting to be able to report to the people of the area that, as a result of exhaustive medical examinations and tests of area water and body fluids and a dental survey of area children, there has been no acceptable evidence to indicate that any sort of human health hazard relative to pollutants exists in the area.

CHAPTER IV

The Effects of Air Pollutants on Soils and Crops

A. RESUME OF ACTIVITIES OF GOVERNMENT DEPARTMENTS RELATIVE TO AGRICULTURAL DAMAGE

308. Upon review of the activities of the Department of Health and the Department of Agriculture and Food with respect to agricultural damage in the Port Maitland area, it was decided to present a chronological sequence of their activities, since both agencies have been deeply involved in this problem. It is only after reviewing the history of legislation regarding air pollution control in Ontario that the involvement of the Department of Health in agricultural matters becomes clear.

309. Since air emissions from industry can cause agricultural damage of many kinds, it was felt by the Ontario Legislature that the Air Pollution Control Service should be actively involved in agricultural areas where potential air pollution might occur. The question of arbitration and negotiation of damages has been included in the *Air Pollution Control Act, 1967*. Although the Department of Agriculture and Food has no specific legislation re responsibility in terms of direct involvement in industrial pollution problems, nevertheless the resources of the various Extension Services of the Department of Agriculture and Food and the Vineland Horticultural Research Institute had been made available to the Port Maitland area farmers. Many of the problems associated with pollution damage to livestock and agricultural crops are new in Canada. Since there is a tremendous literature, as has been pointed out

in paragraph 42, dealing with the problem of fluorides alone, the Committee appreciates the fact that farmers, or indeed agricultural representatives, could not be conversant with this complex problem. Therefore, it is a testament to the interest and efforts of the Department of Health and the Department of Agriculture and Food that many attempts have been made, since 1962, to deal with a problem that offered no easy definition or easy solution. Each has been concerned and each has worked with the other as well as they could reasonably do within the limits of ill-defined responsibilities. Their joint activities in this instance may be summarized as follows:

310. The first indication of injury to agricultural crops received by the Air Pollution Control Service (Department of Health) was in January, 1962, when Mr. W. Fox, Vineland Horticultural Research Station, Department of Agriculture and Food, informed the Department of Health of a letter from Mr. J. Casina, Sr., concerning crop damage. On January 24th, the Air Pollution Control Service agreed to set out sampling stations on Mr. Casina's property. In the summer of 1962, Mr. A. G. Skinner, Agricultural Representative for Haldimand County, advised the Department of Health of injury to tomatoes and corn on the farm of Mr. A. Zynomirski and of strawberry damage on the farm of Mr. J. Casina.

311. An inspection of Mr. Casina's strawberry plants on August 22nd, 1962, by Messrs. Fox, Martin, Dreisinger and Drowley revealed no visible injury; tomato leaf injury was attributed to "flea beetle" by Mr. Fox and Mr. Martin.

312. On September 20th, 1962, Mr. Skinner advised the Air Pollution Control Service that the tomatoes on Mr. Casina's property showed evidence of leaf injury and that the yield was down. On November 19th, Mr. Casina advised the Service that his tomato crop yield for 1962 was 50 per cent of the Ontario average. The Township Council for Moulton Township wrote to the Department of Health on January 17th, 1963, advising that Mr. Casina had requested their assistance in having his crop problems corrected. On January 24th, 1963, the Council was advised that the Air Pollution Control Service could not state the cause of the crop injury and Mr. Casina was notified of this in writing.

313. On July 17th, 1963, a group of governmental officials (Messrs. Fox, Smith and Watson—Department of Agriculture and Food; Mr. Dreisinger—Department of Mines; and Mr. Drowley—Department of Health) inspected several farms in the area.

314. It was generally agreed that very little, if any, injury on Mr. Casina's farm could be attributed to air pollution. On the farm of Mr. L. McIntee, some injury was apparent on lower corn leaves; wild grape leaves had markings characteristic of fluoride damage. No injury was apparent on tomatoes. Severe injury was apparent on gladioli, corn and grapes at the home of Mrs. H. Siddall.

315. The Air Pollution Control Service informed Mr. Casina on November 25th, 1963, that they were not able to "make a definite association between air pollution and crop damage on his farm." This was followed by a meeting at Mr. Casina's farm on December 18th, 1963, where it was explained that a loss of yield could not be correlated with vegetation analyses and lime candle results. The fact that neighbouring farms did not appear to be affected made correlation more difficult. It was also explained that at that time the Service did not have any authority to force industry to correct its emissions.

316. Mr. N. O. Watson, Agricultural Representative, Haldimand County, advised the Department of Health, on April 17th, 1964, that he had visited Mr. Casina on April 16th, 1964. He reported that clover was discoloured and that strawberry plants which were exposed to the atmosphere had a scorched appearance while those which were covered appeared normal. A similar condition was noted at the home of Mr. A. Hoto. Samples of both injured and uninjured plants were taken from both sites by the Air Pollution Control Service. On the basis of examination of visual damage and laboratory analysis, there was a marked similarity of the leaves of plants taken from the Casina and Hoto farms. At the same time, Mr. J. Casina and Mr. A. Zynomirski both claimed that fumes from the area industries were causing retarded growth and major reductions in yield compared to other area growers.

317. Visits were made to both farms by officials of the Department of Agriculture and Food, and the Department of Health. The tomato crop at the Zynomirski farm did not show "expected growth" although some varieties were in better condition than others. Some evidence of disease was present. Injury to apple, pear and shade trees was apparent in the form of blackening of leaf tips and leaf curl.

318. Vegetation at the Casina farm did not show injury comparable to that on the Zynomirski farm. Due to a concern expressed by local farmers, Mr. D. Middleton, Secretary, Ontario Federation of Agriculture, arranged a meeting on July 6th, 1965, which was attended by twelve area farmers. The farmers complained of economic loss due to reduced milk production, lame cattle, and crop

damage. Nine farms were visited by officials of both government agencies and for the first time on an official basis lameness in cattle was observed. Since crop damage was first reported by government officials in 1962, it is incredible that an examination of livestock was delayed so long.

319. ERCO and the O.F.A. were contacted by the Department of Health as to how the problem might be corrected and both parties agreed that they would prefer to have damage claims arbitrated rather than to take the matter to civil court. On August 31st, 1965, ERCO and the O.F.A. agreed to the appointment of Mr. A. G. Skinner as the Arbitrator for the year. This appointment was made by the Department of Health with the concurrence of the Department of Agriculture and Food.

320. All claimants were to submit their claims through the O.F.A. to Mr. Skinner who would base his awards using county averages for any crop damage assessment. Mr. Skinner was free to seek whatever advice he required. Laboratory services would be provided by the Air Pollution Control Service; the Veterinary Services Branch, Department of Agriculture and Food, would provide diagnostic services in relation to livestock. Dr. F. C. Nelson, Head, Veterinary Service Laboratory, Guelph, began his visits to the area at that time. The costs of arbitration services were to be borne by the Department of Health.

321. The awards made by Mr. Skinner were based upon the claims submitted by the individuals concerned. Each statement of award was sent to each claimant, who was advised that, should he find the award unsatisfactory, he would be free to negotiate with the company or take legal action. Thirty-four awards for 1965 (made in 1966) totalled $86,206.49. Of this amount, ten awards were made for 198 head of cattle totalling $38,742.00.

322. Complaints during the growing season of 1966 were again voiced by area farmers. The same system of arbitration was agreed to and Mr. E. McLoughery was appointed Arbitrator for the crop year 1966. His statements of awards were mailed to each claimant on July 14th, 1967.

323. On June 29th, 1966, Mr. A. Rayner, Air Pollution Control Service, in the company of Mr. Ian Smith, Extension Specialist, Vineland Horticultural Station, made a visit to the farms of Mr. R. Elkow, Mr. D. Boorsma and Mr. T. Zynomirski, all of whom had submitted letters in which complaints of damage had been registered. Mr. Elkow remarked that a fifth of his dairy herd was lame and the remainder of the

herd did not seem to be as healthy as usaul. Mr. Boorsma pointed out that of a herd of 53, 12 or 13 cattle were lame. He had been buying his hay over the winter and they seemed to improve slightly (they were then feeding from his own recently cut hay and were getting worse). Mr. Zynomirski indicated that plum, maple, and coniferous trees on his property had suffered damage.

324. Forty-nine awards were made for the crop year 1966, totalling $122,126,00 ($9,903.00 was a carry-over figure from 1965). The 1966 awards included 15 awards for livestock with a value of $37,888.00. These figures included claims for 125 head of cattle, 32 sows, 3 ewes and 170 hens.

325. For the crop year 1967, Mr. Clayton Dean was appointed Arbitrator. The terms of reference were amended to provide as far as possible for assessment on an individual basis rather than on the basis of county averages. Claims submitted for assessment totalled over $180,000.00 (these claims are still being processed by Mr. Dean).

326. During the crop year 1967, in spite of stated improvements within the ERCO plant, complaints from area farmers were made. On June 20th, 26th, and 27th, 1967, Mr. A. Rayner, Chemist, Air Pollution Control Service, in response to these further complaints, visited several farms in the area where agricultural damage was being claimed. He reported on his observations of crop damage on the following farms: McAlonan, Duda, Wright, Karl, Deamude, and Harvey Bros. On August 2nd, 1967, with Mr. K. Best, County Agricultural Representative, he inspected the following farms: McIntee, Rittenhouse, E. Smith and J. Smith. A report on his observations was submitted.

327. In the period between September 26th and October 2nd, 1967, Mr. Howard Henry, Soils and Crops Branch, Department of Agriculture and Food, took a series of 78 vegetation and 76 soil samples from the Port Maitland area. They were submitted to the Air Pollution Laboratory for analysis. The soil samples ranged from a low of 85 p.p.m. to a high of 520 p.p.m. The average reading was 202 p.p.m. These samples were representative of over 34 farms from the area, and showed no obvious pattern or relationship between fluoride content of the soil and the content of the vegetation. During the same period, Mr. H. Henry took 17 samples of hay from 16 different farms. The results ranged from a low of 3 p.p.m. to a high of 191 p.p.m.

328. It would be possible to present the results of all of the sampling data, but instead, comments will be made to indicate the

great difficulty involved in interpreting the results of such samples. Proper interpretations require intensive and rigidly controlled procedures over a lengthy sampling period. The results of sampling did show, however, that on some farms the fluoride concentration of some specific vegetation was high. The fact that they were high does not automatically equate with any prescribed degree of damage. Even in retrospect, it has not been possible to make any overall assessment of the damage pattern of these samples and their fluoride readings. It is hoped that, in subsequent years, farmers complaining of damage will contact ERCO and the appropriate government departments so that damage can be assessed on the spot. ERCO has actually agreed to assess damage settlement on this basis (See Appendix VIII).

B. SOILS AS RELATED TO CROP PRODUCTION

329. The Committee's Consultant on Soils and Soil Productivity was Professor L. R. Webber, Department of Soil Science, University of Guelph.

330. The field work for the soil survey map of Haldimand County was completed in 1935. A map, on a scale of 0.5 inch to 1.0 mile, without report, was published by the Department of Chemistry, O.A.C., in co-operation with the Experimental Farms Service, Dominion Department of Agriculture, Ottawa. Concepts of soil classification and cartographic detail have changed since the release of the Haldimand map.

331. As of January, 1968, no attempt has been made to correlate and update the soils information for Haldimand County. However, the information used in the 1935 soil survey is sufficiently detailed to provide for this study a suitable characterization of the soils in Moulton, Dunn, and Sherbrooke Townships (See Figure 6, which appears on page 130).

332. Each soil type has specific characteristics and, since the soil type determines to a considerable extent the potential productivity, it is necessary to describe the relevant characteristics of the major soil types in Moulton, Dunn, and Sherbrooke Townships as indicated in Table X.

1. Haldimand clay—This is a heavy clay soil which is generally not well drained and is of poor structure and very low permeability. It is difficult to work and to establish good seedbeds and is not responsive to tile drainage. Farmers remove excess water from this type of soil by plowing in 11 to 13 foot lands; it is normally deficient in organic matter, lime and phosphate.

2. Wauseon sandy loam—This is sandy soil with poor natural drainage caused by depressional position or by impermeable clay layer at 1 to 2 foot depths. It is normally a dark-coloured surface soil, is usually difficult to drain, and is normally of low fertility.
3. Caistor loam—Such soil has fair to poor drainage, is normally of low fertility and generally responds to artificial drainage.
4. Berrien sandy loam—This is an undulating deposit of sandy soils over impermeable clay; it is well-drained on the knolls, imperfectly drained in the depressions, and responds to tile drainage if adequate outlets can be established.
5. Granby sandy loam—This is a poorly drained black sandy soil. If drained, it requires large inputs of fertilizer to maintain crop yield. Once drained, this could be a droughty soil in the summer.
6. Bottom land—This is an undifferentiated complex of soil materials adjoining stream courses. It is subject to flooding and is normally used as unimproved pasture land.

TABLE X

APPROXIMATE ACREAGES OF THE MAJOR SOILS OF DUNN, MOULTON, AND SHERBROOKE TOWNSHIPS*

	Acres	Main Soils Present and Acreage			
1.0-mile radius					
Dunn	588	Haldimand	588		
Sherbrooke	512	Caistor	307	Bottom Land	205
Total	1,100				
2.5-mile radius					
Dunn	3,967	Caistor	2,867	Haldimand	1,715
Sherbrooke	2,432	Bottom Land	1,178	Oneida	870
Moulton	1,178				
Total	7,577				
5.0-mile radius					
Dunn	10,740	Caistor	6,280	Haldimand	6,060
Sherbrooke	5,130	Wauseon	5,095	Berrien	3,870
Moulton	12,140	Granby	2,710	Bottom Land	2,050
Canborough	205				
Total	28,215				

*To obtain data arcs were drawn with centre on inner lighthouse at Port Maitland Harbour (See Figure 6).

333. Soil productivity is the capacity of a soil for producing a specified crop or sequence of crops under a defined system of management. Productivity is measured in terms of outputs, e.g., crop yields, pounds of meat per acre, pasture, number of cow-days pasture, and in terms of inputs of production factors, e.g., fertilizer, tillage, drainage, irrigation, crop variety. By assessing the success of output against what has been put into the soil, a measure of productivity can be gained.

334. Soil fertility is that quality of soil that enables it to provide compounds in adequate amounts and in proper balance for the growth of specified crops.

335. For 12 years, the Department of Soil Science of the Ontario Agricultural College operated a Regional Research Station about one mile NE of Rainham in South Cayuga Township. The soil type was the Haldimand clay. The following observations and comments regarding the soils in Haldimand County are based on research findings from the station together with knowledge gained of the soils in the county not found on the station. Management experiements, including investigation of tillage, fertilization, crop rotation, liming and soil conditioners, were conducted. The results indicated that outputs from the Haldimand clay were restricted primarily by inadequate drainage and low levels of soil fertility. The physical condition of the Haldimand clay restricts the effectiveness of tile drainage. County demonstrations have indicated that tile drainage produced no significant improvement in crop yields. Outputs, quantitatively and qualitatively, are primarily influenced by the system of management that a farmer or operator follows. Management encompasses the various aspects of choice of crop and variety, fertility and crop residue management, timeliness in tillage, planting and harvesting, weeds, insect and disease control, pasture management, drainage and irrigation, and the use of soil amendments.

336. In parts of Haldimand County, soil productivity is impaired by inadequate soil drainage. This is the case in the Haldimand-Caistor series or where coarse to medium textured soils are underlain by impervious clay strata as in the Berrien, Wauseon, and Granby soils. It is also true in the topographic locations, particularly the muck soils where depressions do not have adequate natural drainage outlets.

337. Management does not normally change texture but does influence structure. Improved soil structure to aid aeration and drainage may be accomplished by additions of organic material in man-

ures or through root accumulations accompanying higher crop yields. An investment in tile drainage on responsive soils normally returns a greater profit than a similar investment in fertilizer, amendments and biocides. The exceptions are in soils with unique soil physical limitations as in the Haldimand and Caistor soils.

338. To offset the natural limitations that occur in the soils in Moulton, Sherbrooke, and Dunn townships, a farmer must practice good soil management. Farmers differ in their abilities to interpret, understand, and implement soil management recommendations. This is reflected in farm appearance, income and in the quantity and quality of crops and livestock produced.

339. Most of the soils in the three townships require some form of artificial drainage. When the drainage is improved, the soil may be prepared properly and early in the spring. Drainage increases the range of crops and often results in an increase in farm income. Tile drainage would be effective on most of the soils but not on the Haldimand clay. An essential part of a tile drainage system is an adequate outlet. Farmers in the area have found that plowing in narrow lands, 11 to 13 feet, enhances the removal of surplus water.

340. Lime should be added to soils if the pH is 6.0 or less. Where legumes are desired in the hay or pasture mixture, lime should be added if the pH is 6.2 or less. Modern farming dictates that a farmer should add the major plant nutrients, nitrogen, phosphorus, potassium, and perhaps micronutrients such as boron, if crop production is to be maintained at the highest economic level.

C. EFFECTS OF FLUORIDES ON SOILS

341. It is naturally of great concern to know whether or not fluoride deposited on the soil, in any of its combined forms from industrial plant emissions, has a deleterious effect upon the soil and its productivity and indeed subsequently on plants which are grown on such soil.

342. The natural level of fluoride in soils varies with the type of soil. Robinson and Edgington (1946)[37] analyzed the fluoride content of 30 soil profiles varying in texture, parent material, and geographical location in the United States. They found that the fluoride content varied from a very insignificant amount to over 7,000 p.p.m. The average for surface soils was 292 p.p.m. The main source of the fluoride was found to be the micaceous clays. Soils containing phosphate rock would have a high fluoride content.

343. Although there are no comparable soil analyses for fluoride content of Ontario soils, Drowley and his associates, in 1963, reported that a field-tile manufacturing plant in the Niagara Peninsula was using a clay which contained 368 p.p.m. of fluoride[38]. As a result of a study by Webber and Shivas (1953) and others, it is known that in many Ontario soils the content of hydrous mica is high. The occurrence of various micas in Ontario soils was reported later by Dell (1959)[3]. It should be pointed out that fluoride content of micaceous clays is high, samples from Wisconsin showing up to 5,800 p.p.m. It would seem logical then to deduce from such analyses and the high known fluoride content of micaceous clays that the fluoride content of fine textured soils in Ontario would probably equal the average value of 292 p.p.m. for the 30 American soils studied by Robinson and Edgington[37].

344. MacIntire (1949)[39] found that fluoride contained in compounds added to the soils in lysimeters* was retained almost completely by the soil. However, when quenched calcium silicate slag (from electric furnaces used to reduce phosphate rock) containing 4 per cent fluoride was applied to soils, the fluoride content of the percolate was significant.

345. Even after 10 years of leaching with rain water, certain soils systems retained 98 per cent of the fluorine added in 2½ tons of calcium fluoride per acre. This input of fluorine exerted no ill effects upon the soil or the vegetation which grew on it (Sphecht and MacIntire, 1961)[40].

346. Since apatite has a very low solubility in soil, the behaviour of the fluoride component of phosphate rock was such as not to contribute significantly to the fluoride content of percolates through the soil (MacIntire et al, 1954)[41].

347. The predominant natural sources of fluoride in soils are associated with the content of apatite and micaceous minerals. The solubility of these minerals is extremely low, hence they are unlikely sources of fluoride in forms available to plants.

348. Although fluoride is added to soils through the use of chemical fertilizers, particularly superphosphates, the calcium fluoride content of large inputs of superphosphate has had no adverse effect on crops and has not caused appreciable increases in the fluoride content of the vegetation (MacIntire, 1957)[42].

*A lysimeter is a quantity of soil retained by a metal cylinder equipped with a device to collect all the water that goes through the soil.

349. Fluoride may be added to soils by industrial emission. If an industrial plant were to release 10 tons of elemental fluoride in the air per day and half of this settled within a ten-mile radius of the plant, the average rate of fluoride addition to the surface 6 inches of soil would be about 10 p.p.m. per year (Hansen et al, 1958)[8].

350. In September, 1967, Mr. Henry, Soils and Crops Branch of the Ontario Department of Agriculture and Food, as mentioned in paragraph 327, took 76 soil samples from several farms in the Port Maitland area. These samples were analyzed for their fluoride content by the Ontario Air Pollution Laboratory. The fluoride content varied from 85 to 520 p.p.m., with an average of 202. On one farm, two samples from two different fields showed values of 190 and 300 p.p.m. F; on another farm, 155 and 235 p.p.m., and on another, values of 220 and 450 p.p.m. were found. Such levels are well within the range of the fluoride content of agricultural soils.

351. Certain soil mechanisms immobilize the addition of fluoride to a soil. These may be summarized as follows:

(a) Fluoride in soluble forms, such as NaF, KF, HF, and H_2SiF_6 are fixed by reactions with the calcium compounds in a soil. The low solubility of CaF_2 prevents the uptake of fluoride by plants and its appearance in leachates.

(b) In acid soils, particularly sandy-textured ones that are low in calicum, the fluoride is fixed by the common hydroxyl-containing minerals; the hydroxyl ion (OH) is released when fluoride is added. There is evidence that an exchange occurs with the hydroxyl ion ($Al(OH)_3$ aluminum hydroxide). Bower & Hatcher (1967)[43] proposed that waters from deep aquifers in the Western United States containing 5 to 15 p.p.m. of fluoride could be "defluorinated" for municipal use by causing the water to percolate through the soil.

(c) The organic and inorganic colloidal fractions of soils also tend to act as fixation agents in respect to added fluoride, preventing uptake by plants.

352. Fluorides reaching soils, through being washed off vegetation by rain, are in the acidic forms or their salts. The acidic forms react with the supply of limestone, dolomite or silicate in soils to form calcium fluoride, magnesium fluoride or calcium silicofluoride. In soils well supplied with calcium, the added hydro-

fluoric acid reacts and develops calcium fluoride; hydrofluosilicic acid forms calcium silicofluoride. Neither of these fluorides has proven toxic to plants, in spite of the tendency for fluoride to occur in the leachates where soils had been treated with quenched slag for the rock phosphate treating process. Apparently, there was no increase in the uptake of fluoride by vegetation from the slag-treated plots (MacIntire, 1957)[42].

353. Hansen and associates (1958)[8] showed that the naturally occurring fluoride content of soils was related to the contents of clay, organic matter and lime, as shown in the following table:

TABLE XI

CHEMICAL ANALYSIS OF UTAH SOILS

Item	*Orem Loamy Sand*	*Ironton Loam*	*Mendon Silt Loam*	*Petersboro Silt Loam*	*Taylorsville fine sandy Loam*
pH	8.0	8.5	7.4	7.4	7.8
Organic matter (%)	1.2	11.3	4.0	5.5	4.5
Clay (2.ou) (%)	11.0	20.0	24.0	18.0	25.0
$CaCo_3$ (Lime) (%)	9.36	52.0	0.03	10.5	17.0
Fluoride p.p.m.	248.0	874.0	810.0	368.0	490.0

354. Samples of the Orem and Ironton soils were taken from the areas where atmospheric fluorides have been high for a number of years. The fluoride levels of alfalfa and turnips grown on these high fluoride-containing soils but in an atmosphere low in fluorides were less than 20 p.p.m., or within the range of fluoride content of crops grown normally in areas free of atmospheric fluoride.

355. In Florida, the fluoride content of Leon fine sand, pH 4.1, was 35 p.p.m. This soil was obtained from an area two to five miles from a plant grinding and processing rock phosphate to manufacture triple superphosphate (Sphecht and MacIntire, 1961)[40]. The authors concluded that grass took up added fluoride from the acidic Leon soil within the pH range of 4.1 to 4.5.

356. Hurd-Karrer (1950)[44] studied the absorption of fluoride by collards and buckwheat from a sassafras loamy sand having a

pH near 5.0 and containing 12 p.p.m. of naturally occurring fluoride. Half of these plots were limed to pH 7.5 before seeds were planted. The fluoride content of injured collard plants on unlimed soil was 96 p.p.m. On the limed soil, the plants were uninjured with a fluoride content of 43 p.p.m. in the plant tissue.

357. Prince and his colleagues (1949)[45] found that tomatoes and buckwheat were injured by the uptake of added fluorine from the acid sassafras soils. Toxicity, as reflected in yields, was reduced by increasing the soil phosphate content and by raising the pH from 5.5 to 6.5. The level at which fluoride became toxic varied with soil type, lime, pH and phosphate content and the type of plant grown. The experiments suggest that:

(a) The total fluoride content of a soil does not indicate the fluoride content of plants grown on that soil; one must distinguish between available and total fluorides.

(b) Corrective measures to overcome fluoride toxicity appear to be related to soil pH, lime and phosphate content.

(c) Fluoride from air pollutants is absorbed by or deposited on the leaves rather than absorbed from the soil. Fluoride contamination of vegetation occurs directly from the air. After the fluoride reaches the soil, it is apparently inactivated and absorption by roots is negligible.

D. EFFECTS OF FLUORIDES ON PLANTS

358. This section of the report, taken from Professor Tanner's prepared presentation, deals primarily with generally accepted concepts on the effects of fluorides on plants.

359. As indicated previously, fluoride is generally present in the soil to the extent of several hundred p.p.m. Plants show little tendency to take it up and the amount taken up from the soil by plants is usually unrelated to the fluoride content of the soil. Soil type, calcium and phosphorus levels, and pH appear to be the controlling factors. When injury does occur, the symptoms are almost identical to those produced by exposing the leaves to hydrogen fluoride (HF) gas. Absorption by the roots from the soil is usually indicated by abnormally high concentrations of fluoride in the roots relative to the above ground portions of the plant. Liming soils to pH 6.5 will insure almost complete fixation of the soluble fluoride compounds present or added to the soil.

360. With few exceptions, the fluoride content of plants not exposed to atmospheric fluoride is in the range of 2 to 20 p.p.m. Not-

able exceptions include camellia leaves (1,900 p.p.m.), tea leaves (up to 800 p.p.m.) and buckwheat (196 p.p.m.).

361. Most cases of fluoride injury to plants have occurred as a result of atmospheric pollution—particulate and gaseous. Particulate fluoride is, as implied in the name, visible and near-visible particles of fluoride-containing compounds. The fluoride pollution problem has two principal aspects as it relates to plants: first, direct effects on crop and ornamental plants as it relates to injury and yield reduction; second, raising the fluoride level in or on foliage to the level which causes fluorosis in consuming animals. The particulate fluorides contribute to the latter aspect but not appreciably to the former, since they are not believed to enter the plant via the leaf to any significant extent. Gaseous forms of fluoride, including hydrogen fluoride (HF) and silicon tetrafluoride (SiF_4), contribute to both aspects.

362. Gaseous fluorides enter the plant primarily through the leaf stomates without causing injury at the point of entry. There does not seem to be any correlation between stomates per unit leaf area and sensitivity of a species. Leaf stomates are microscopic "pores" on the leaf surface through which water is lost from the plant by transpiration, and carbon dioxide (CO_2) enters the plant when photosynthesis is proceeding. Stomates are present on both the upper and lower surface of the leaf of most species. Each stomate is surrounded by two specialized cells, called guard cells, whose movements, due to changes in turgidity and light, govern the opening and closing of the pore. Gaseous materials move in and out, depending upon their relative concentrations inside and outside of the leaf. For example, during daylight, CO_2 moves into a leaf as the plant using it forms sugar.

This continued use of CO_2 (in light) insures that there is always more outside than inside the stomate, hence CO_2 continues to move in. Gaseous HF acts in a similar manner. In most species, the stomates are closed at night; hence, plants tend to suffer less damage in the dark than in the light. As indicated previously, during daylight hours the wind direction in the Port Maitland area occurred 47 per cent of the time from the SW; that is, the period during the day when the vegetation was most susceptible coincided with the period during the day when the predominant wind direction was such that pollutants would be present.

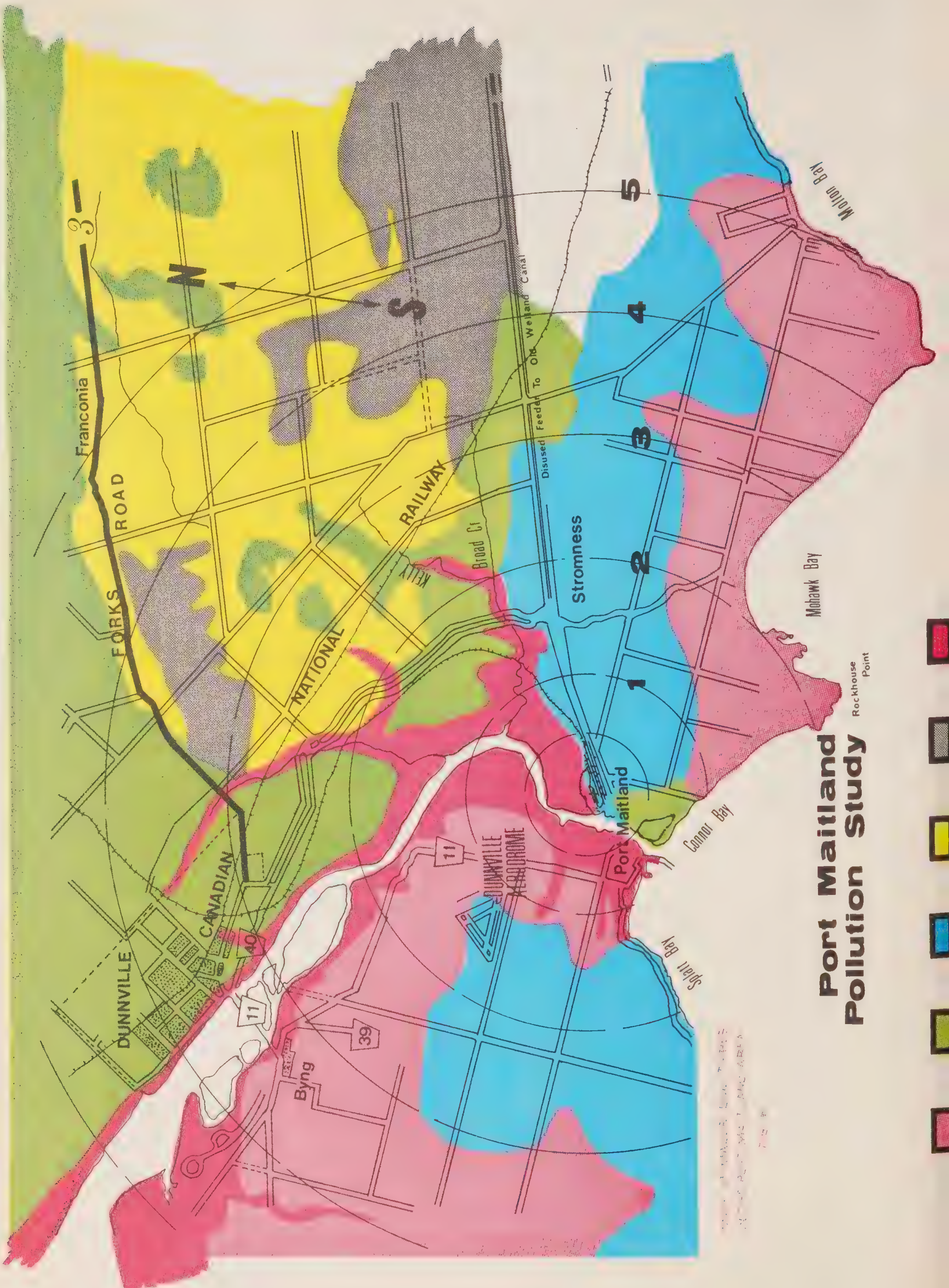
363. Environmental conditions which favour good growth, i.e., warm, bright, humid days, tend to be conducive to stomate opening and therefore to increase fluoride injury. It is generally recognized that land areas adjacent to large bodies of water, such as the Port

Maitland area, usually have higher humidities during daylight hours, due to the lake breeze effect, than at night. This, too, would increase the likelihood of pollution injury to vegetation.

TABLE XII

PORT MAITLAND AREA PRECIPITATION DISTRIBUTION (1961-1967)

Year	*Dry Periods*	*Amount*	*Wet Periods*	*Amount*	*Other Comments*
1967	——		May 1-15	(3.0″)	Fairly even distribution during remainder of the season.
	July	(0.9″)	Aug. 2-19	(6.8″)	
	Sep. 1-20	(0.3″)	——		
1966	——		Apr. 23-27	(1.5″)	The long dry period in late June-early July caused corn to wilt for 2 to 3 weeks in the area.
	May 19-June 8	(0.2″)	——		
	June 17-July 17	(0.2″)	——		
	Sep. 5-19	(0.2″)	——		
	Oct. 1-31	(1.2″)	——		
1965	May 7-24	(0.3″)	——		Fairly even distribution during remainder of the season.
	June 3-15	(T)	——		
	July 10-31	(0.2″)	——		
	Sep. 11-Oct. 6	(0.4″)	——		
1964	——		Apr. 1-30	(5.2″)	Precipitation slightly below normal in June.
	——		May 8-14	(2.8″)	
	——		July 11-14	(2.5″)	
	July 15-31	(T)	Aug. 11-22	(6.3″)	
	Aug. 23-Sep. 18	(0.1″)	——		
	Sep. 19-Oct. 31	(1.4″)	——		
	Nov. 18	(1.6″)	——		
1963	Apr. 1-16	(0.3″)	Apr. 17-19	(1.7″)	Precipitation distribution near normal in May and late July.
	May 28-June 8	(0.1″)	——		
	June 11-26	(0.1″)	——		
	June 28-July 13	(0)	Aug. 7-13	(4.3″)	
	Aug. 17-27	(0.1″)	——		
	Sep. & Oct.	(1.5″)	——		
1962	May 9-June 9	(0.7″)	June 11-13	(2.6″)	Fairly even distribution during remainder of the season.
	June 14-July 19	(0.4″)	July 20-25	(2.3″)	
	——		Aug. 4-8	(2.4″)	
	——		Sep. 27-28	(2.2″)	
1961	——		Apr.1-30	(6.0″)	Precipitation distribution adequate during May and June.
	July 3-12	(0.1″)	July 30-Aug. 6	(4.5″)	
	Sep. 2-22	(0.2″)	——		
	Sep. 26-Nov. 11	(0.8″)	——		


Port Maitland
Pollution Study
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FORKS ROAD
CANADIAN NATIONAL RAILWAY
DUNNVILLE
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Broad Cr
Disused Feeder To Old Welland Canal
Stromness
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4
5
Molton Bay
Mohawk Bay
Rockhouse Point
Connor Bay
Port Maitland
DUNNVILLE AERODROME
Sprau Bay
Byng
11
39
40
3

364. Distribution and amount of rainfall are very significant factors related to the effects of air pollutants on plants. Adequate to ideal moisture results in conditions conducive to rapid growth, open stomates, and increased susceptibility. Frequent rains tend to wash particulate fluorides off the leaves into the soil. Dry periods result in accumulations of particulate fluorides on the leaf, but less entry of gaseous fluorides through the stomates. Dry periods also reduce injury by stimulating the development of increased resistance (hardening) of the plants. An excess or deficiency of nutrients results in less gaseous fluoride absorption and injury than medium levels of nutrients. The precipitation distribution for the Port Maitland area (1961-1967) is shown on Table XII.

365. Hydrogen fluoride may be absorbed on the outside of the leaf, and may be washed off by rain, volatilized or absorbed into the leaf. The gaseous fluorides absorbed by the leaf through the stomates are moved (translocated) toward the leaf margins in broad leaf plants and toward the tips in leaf blades. By this means the body of the leaf, where absorption takes place, may remain at a low level of concentration while the margins build up to lethal concentrations causing marginal burning (necrosis). By this mechanism plants can accumulate fluoride in quite significant amounts over a period of time. For example, it has been observed that over a growing season a plant exposed to an atmospheric concentration of 5 parts per billion (p.p.b.) accumulated fluoride in the plant to a concentration of 500 p.p.b.

366. It is of extreme practical significance that fluorides absorbed via the leaf tend to remain in the leaf. There is little evidence to indicate that significant amounts move into the reproductive organs even when the leaves contain very high levels. So, while the leaves might contain fluoride in amounts that would be prohibitive for feeding, the fruit or grain could be harvested with little chance of contamination. While damage to fruits has been reported, this appears to be due primarily to direct atmospheric effects rather than to what is translocated from leaves.

367. Some plants have the ability to precipitate out the fluoride as insoluble salts in their tissue. This reduces the toxic effects but assists in maintaining a gradient which results in concentration build-up. Other plants may form organic fluoride compounds such as fluorooleic and fluoropalmitic acids.

368. Recent research indicates that leaves may lose significant amounts of fluoride back into the atmosphere. This presumably could occur only after the concentration in the air was reduced. The

mechanism whereby fluoride can be fixed (stored) temporarily and evolved back into the air is not understood. There is also some evidence which indicates that this loss from the leaf can occur after a plant is cut for hay.

369. It is of considerable interest and biological significance that there is a wide variation in the susceptibility of different plants to fluoride injury. This variability exists:

(i) between species
(ii) within species
(iii) within a variety depending on the environmental conditions and the physiological condition of the plant.

370. This relative sensitivity of plants to hydrogen fluoride is indicated in Table XIII[46].

371. It is of particular interest to note in Table XIII, as Professor Tanner pointed out, that there are some plants which may be classified as "intermediate" in their resistance and some as "resistant" to fluoride, when plants are classified on their relative degree of sensitivity. In the same way the Table shows that one type of pine may be sensitive while other types are resistant. And would it not be the case that dandelion, plantain and ragweed are in the resistant group?

372. It has also been noted that the species most susceptible to HF injury (Jerusalem cherry, gladiolus, tulip, one type of corn, and sorghum) were resistant to sulphur dioxide. High concentrations of HF, even on quite resistant species (alfalfa, barley, cotton) may cause a temporary reduction in the rate of photosynthesis until the fluoride can be moved to the margin by translocation. There appears to be a threshold for each species below which there is no interference with photosynthesis.

373. While it has been reported that the reduction in photosynthesis varies directly with leaf area damaged, this has been shown only on individual space-planted plants. The relationship probably does not hold in a closed canopy of a community of plants. Unless visible injury occurs, no damage has been done; that is, there is no such thing as invisible injury. This is in contrast to crop yield losses due to nutrient deficiencies which may occur without visible injury.

374. Foliar symptoms of fluoride injury vary somewhat from species to species, but fall into two general types—marginal necrosis (sometimes referred to as tip burning, scorching or lesions)

TABLE XIII

RELATIVE SENSITIVITY OF CULTIVATED AND NATIVE PLANTS TO INJURY BY HYDROGEN FLUORIDE (7-9 DAYS)

CULTIVATED PLANTS

Sensitive
0.005 p.p.m.

Class 1

Gladiolus	ABC
Pine (young needles)	B
Apricot (Chinese)	BR
Azalea	C
Blueberry (Jersey)	C
Prune (Italian PRH 1)	C
Tulip	B
Jerusalem cherry	B

Class 2

Milo Maize	B
Corn (Dixie 17, Funk 134 & 512)	B
Corn (Spancross)	B
Corn (bantam)	A
Sweet potato (Triumph)	B
Apricot (Moorpark)	ABC
Prune (late Italian)	AB
Peach (some varieties under best conditions)	B
Peach (Elberta, Lovell stock)	C
Strawberry (Marshall)	C
Grapes (some European varieties)	RW
Iris	AB

Intermediate
0.005-0.010 p.p.m.

Class 3

Corn (Golden Cross bantam)	B
Pepper (California Wonder)	C
Raspberry (Washington)	C
Aster & Sweet William	A
Dahlia (Immature leaves)	A
Dahlia	C
Petunia petals	A
Clover	B
Barley, flax, oats & rye (young plants)	A
Apple (Delicious) Cherry (Bing)	C
Apple	B
Birch, hawthorne, silver maple, mountain ash, mulberry, sycamore & yellow willow	C
Peach	AB
Iris & begonia	B

Class 4
0.01 p.p.m.

Azalea	BC
Begonia (some varieties)	B
Rose, geranium, coleus, lilac, spirea	B

Resistant
0.01 p.p.m.

Class 5

Columbines & Canterbury Bell	A
Dogwood & lilac	C
Lobella & petunia	A
Rose	B
Apple (red Jonathan)	C
Live oak & pine	B
Tomato	ABC

Class 6

Alfalfa (Ranger)	C
Corn (Golden cross bantam)	C
Cotton	A
Tobacco	BC
Bean & celery	B
Cucumber & squash	B
Squash (Hubbard & Zucchini)	AC
Cabbage, cauliflower, eggplant, onion, pepper & soybean	A
Parsnips (hollow crown)	C
Tomato (Marglobe)	C
Chrysanthemum	AB
Marigold, privet & snapdragon	A
Sweet pea (Spencer hybrid)	C
Rhododendron (Pink Pearl)	C

TABLE XIII (cont'd)

RELATIVE SENSITIVITY OF CULTIVATED AND NATIVE PLANTS TO INJURY BY HYDROGEN FLUORIDE (7-9 DAYS)

NATIVE PLANTS

Sensitive 0.005 p.p.m.		*Intermediate* 0.005-0.010 p.p.m.		*Resistant* 0.01 p.p.m.	
Class 1		Class 3		Class 5	
Pine (ponderosa, young needles)	BC	Pokeweed, crabgrass	B	Pigweed, dock	B
Larch	C	Smartweed, Johnson grass	B	Carpet weed	B
Hypericum	B	Oxalis	B	Pine (lodgepole)	C
		Chickweed, barnyard grass	B	Pine (ponderosa, old needles)	C
Class 2		Class 4		Class 6	
Crabgrass	B	Pokeweed, pigweed, lamb quarters, dock, plantain	B	Dandelion, plantain, purslane, galinsoga, bidens, night-shade & ragweed	B
Cattail	B			Douglas fir, grand fir, hemlock, white pine, Engelmann spruce	C
Johnson grass, oxalis, smartweed, buckwheat	B				
Pine (ponderosa, 3-4 month old needles)	C				

Sources:
A=American Smelting and Refining Company; B=Boyce Thompson Institute;
C=State College, Washington; R=B. L. Richards; W=F. W. Went

and interveinal chlorosis. Marginal necrosis is the most common symptom of fluoride toxicity and has been observed on many domestic plants including plum, cherry, oak, apricot, peach, walnut, lilac, rose, beet, tomato, pine, grape, gladiolus, strawberry and other crops. With many species, the marginal necrosis is preceded by the appearance of grey or light green water-soaked lesions which later turn tan or whitish-brown. Injury usually appears first at the margin or tip and moves inward and/or downward until a large part of the leaf is affected. For example, in gladiolus, a plant very sensitive to fluoride, injury starts at the tip of the blade and gradually extends downward maintaining a fairly uniform front which is sharply separated from the healthy area by a very narrow band of chlorotic (yellow-green) tissue, often streaked with red.

375. Interveinal chlorosis, which sometimes resembles manganese deficiency, frequently results from exposure to low concentrations of fluoride for sustained periods. This is considered to be a symptom of a chronic pollution problem. Acute damage in the form of interveinal and marginal bleached areas, similar to sulphur dioxide injury, may occur after short-term exposure to high concentrations. Although fluoride toxicity symptoms are relatively characteristic, a number of other factors such as other pollutants, excessive soil salts, extreme moisture stress and mineral deficiencies, will produce similar symptoms. For this reason visual diagnosis must be confirmed by chemical analysis of leaves.

376. Many workers have quoted atmospheric concentrations which have caused injury to plants. The value of the figures reported is doubtful since the time of exposure and other conditions of the experiment are often not clearly stated. Further, it appears to be very difficult to maintain a constant concentration of HF in a fumigation chamber. In any case, these figures give little indication of what concentrations might be required to damage plants subjected to pollution in the open, especially as the effect of fluctuating emissions and variations in other environmental factors do not appear to have been studied.

377. The reasons for plants varying in sensitivity are not known, nor is the actual physiological-biochemical mechanism of toxicity known. With few exceptions, the more resistant species and varieties tend to accumulate more fluoride than the susceptible varieties. While accumulation of fluoride within the plant tends to follow the chlorotic pattern, chlorosis and fluoride content are not quantitatively correlated to each other. It is not uncommon for a healthy appearing leaf to contain as much fluoride as a leaf

showing symptoms. Leaves contain more fluoride than stems and stems more fluoride than fruit. Older leaves contain more fluoride than younger ones. A young, fully developed leaf is more sensitive to HF fumigation than older or immature leaves.

378. Viewed under a microscope, the HF damage to leaf cells cannot be distinguished from SO_2 damage. The biochemical-physiological basis of the histological changes which take place are not clear. The effect of fluoride on the activity of many enzymes and numerous metabolic reactions has been studied and the literature is voluminous in this area. At the present state of knowledge, the significance of a change in one metabolic step for the physiology of the plant as a whole, and the relation of metabolic changes in injury or reduced yield, cannot be adequately determined.

379. In addition to leaf injury, other plant responses have been observed. These include observations on new shoots, which may show poor development when submitted to prolonged exposure of HF at a few parts per billion; effects on petals which tend to be more resistant than leaves; local softening of fruit in plums, pears and peaches, and some loss of crop due to poor seed set in some crops when exposed at pollination time.

E. EFFECT OF SULPHUR DIOXIDE ON SOILS

380. The sulphur content of soils is about 0.05 per cent (S) or 1,000 pounds per acre-furrow slice. In surface soils, sulphur occurs primarily in the organic matter. Sulphur is added to soils by atmospheric fallout, animal manures, and chemical fertilizers.

381. Sulphur is an essential element for the growth of plants and is absorbed by plants almost entirely as an inorganic sulphate (SO_4).

382. When sulphur dioxide is absorbed by a soil, it is ultimately oxidized to sulphuric acid by soil bacteria. The acid in turn reacts with the soil bases to form sulphates. The reactions yield sulphates which may be used by the plants or leached out of the soil. It is possible that the soil would become acidic if the additions of sulphur are continued until most of the bases have reacted with the acid.

383. To prevent the development of very acidic soils by the absorption of sulphur dioxide, it is necessary that a farmer maintain the soil pH above 6.0 by adding lime. By maintaining such a minimum soil acidity level, SO_2 accumulation in the soil is rendered relatively harmless and therefore the SO_2 will have no influence on plants grown on such soils.

F. EFFECTS OF SULPHUR DIOXIDE AND ZINC ON PLANTS

384. Sulphur dioxide (SO_2) has been studied longer and more extensively than any other air pollutant. Its effects on plants are fairly well established and the methods of detecting its presence are precise and predictive of its effects on plants.

385. Sulphur is one of the sixteen elements essential for plant growth. It is a constituent of certain amino acids, which are the structural units of proteins. Sulphur is also present in other compounds which are essential in many of the oxidation-reduction reactions which occur in living systems. Sulphur is taken up from the soil in the form of sulphate (SO_4) and is reduced to the sulphydryl form for incorporation into protein.

386. The SO_2 gas, when present, enters the plant via the stomates in the same manner and under the same conditions as CO_2 and HF gain entrance. The gas is absorbed into the mesophyll cells of the leaf. Toxicity is largely due to the reducing properties of the gas. Different species of plants vary over a considerable range in their susceptibility to injury by SO_2. These differences seem to be due primarily to differences in the rate of absorption of the gas by the leaves. Plants with succulent leaves of high physiological activity are generally very sensitive to SO_2. These include alfalfa, small grains, squash and grapes; corn is an exception. Vegetation is most susceptible during the months of active growth (June and July) and during periods of high relative humidity. At other times, as during May, August, September and October, plants frequently survive exposure to several times the threshold concentrations of SO_2 for injury.

387. Table XIV indicates the degree of sensitivity of many crops relative to alfalfa which is highly sensitive[57]. Alfalfa shows injury after one hour when exposed to 1.25 p.p.m. of SO_2. (The values presented in the table were obtained under carefully controlled experimental conditions, hence should be used with caution when thinking in terms of farm-raised crops). Some discrepancies do exist between the lists published by various investigators and, recently, it has been shown that concentration has a greater effect than time. A given amount spread over a long time is not as damaging as the same amount over a short time. A recent report states that the concentration-time variables are insufficient criteria for assessing or predicting injury, indicating that frequency and duration of peak and recovery periods can greatly modify the effects.

388. Many attempts have been made to show evidence of "hidden injury." However, no reduction, in yields of plants ex-

TABLE XIV

RELATIVE SENSITIVITY OF CULTIVATED AND NATIVE PLANTS TO INJURY BY SULPHUR DIOXIDE

Sensitive		*Intermediate*		*Resistant*	
Alfalfa	1.0a	Cauliflower	1.6a	Gladiolus (1.1-4.0)b	2.6a
Barley	1.0	Parsley	1.6	Horseradish	2.6
Endive	1.0	Sugar beet	1.6	Sweet cherry	2.6
Cotton	1.0	Sweet William	1.6	Canna	2.6
Four o'clock	1.1	Aster	1.6	Rose	2.8-4.3
Cosmos	1.1	Tomato (1.3-1.7)b	1.7	Potato (Irish)	3.0
Rhubarb	1.1	Eggplant	1.7	Castor bean	3.2
Sweet pea	1.1	Parsnip	1.7	Maple	3.3
Radish	1.2	Apple	1.8	Boxelder	3.3
Verbena	1.2	Catalpa	1.9	Wisteria	3.3
Lettuce	1.2	Cabbage	2.0	Mock orange	3.5
Sweet Potato	1.2	Hollyhock	2.1	Honeysuckle	3.5
Spinach	1.2	Peas	2.1	Hibiscus	3.7
Bean	1.1-1.5	Gooseberry	2.1	Virginia creeper	3.8
Broccoli	1.3	Zinnia (1.2)b	2.1	Onion	3.8
Brussels Sprouts	1.3	Marigold	2.1	Lilac	4.0
Pumpkin	1.3	Hydrangea	2.2	Corn	4.0
Table beet	1.3	Leek	2.2	Cucumber	4.2
Oats	1.3	Begonia	2.2	Gourd	5.2
Bachelor's button	1.4	Rye (1.0)b	2.3	Chryanthemum	5.3-7.3
Clover	1.4	Grape	2.2-3.0	Snowball	5.8
Squash (1.1-1.4)b	1.4	Linden	2.3	Celery	6.4
Carrot	1.5	Peach	2.3	Citrus	6.5-6.9
Swiss chard	1.5	Apricot	2.3	Cantaloupe (Muskmelon)	7.7
Wheat	1.5	Nasturtium	2.3	Arbor vitae	7.8
Gaura (1.0)b	1.0	Elm	2.4	Current blossom	12.0
Tobacco tree (N. glauca)	1.1	Birch	2.4	Live Oak	14.0
June grass (B. tectorum)	1.0	Iris	2.4	Privet	15.0
Prickly lettuce	1.0	Plum	2.5	Corn silks & tassels	21.0
Mallow	1.1	Poplar	2.5	Apple blossoms	25.0
Ragweed	1.1-1.2	Dandelion	1.6	Apple buds	87.0
Curly dock	1.2	Orchard grass	1.6	Purslane	2.6
Bouncing bet	1.3	Rouchpigweed (Redroot)	1.7	Sumac	2.8
Plantain	1.3	Black mustard	1.7	Shepherd's purse	3.0
Sunflower	1.3-1.4	Smartweed	1.8	Milkweed	4.6
Rye grass	1.4	Lamb's quarters	1.8	Salt grass	4.6
		Sweet clover	1.9	Pine c	7-15.0
		Nightshade	2.0		
		Hedge mustard	2.1		
		Cocklebur	2.3		
		Tumbling mustard	2.4		

a: Factors of relative resistance compared with alfalfa as unity.

b: More probable factors based on later experience.

c: Data for pine represent October fumigations in Palo Alto, California.

posed to SO_2, could be demonstrated unless the leaves were visibly injured. Similarly, fumigations which do not cause leaf injury do not reduce photosynthesis significantly, although temporary reductions in photosynthetic rates have been observed. Exposure to SO_2 levels insufficient to produce visible symptoms in plants may, in fact, increase yields on many soils.

389. Reduction in yield is reported to be proportional to leaf area destroyed; however, this relationship would not be direct in a crop production system.

390. As indicated above, the concentration-time relationship is most significant and Table XIV is presented only as a possible guide; the data for the short-term controlled exposures may be at variance with data obtained from long-term studies under conditions of actual exposure to industrial SO_2 emissions. The Committee does not have such data available at the present time.

391. Sulphur dioxide causes two types of injury on leaves of plants — acute and chronic. The acute markings are marginal and/or interveinal collapse areas, usually of an ivory colour, though in some species they may be brown to reddish-brown. The chronic markings are areas of limited cell injury but not of total collapse. They range in colour from brownish-red to yellow to white. Chronic injury results from fumigation treatments of insufficient intensity to cause acute markings, or from prolonged exposure to low concentrations which eventually cause sulphate toxicity. Determination of the sulphur content of leaves will usually confirm exposure to SO_2.

392. Injury from SO_2 is local; injured areas of the leaves never recover but, since no systemic effects have taken place, the uninjured areas and new leaves develop normally. Certain diseases and environmental conditions may cause symptoms similar to those caused by SO_2. Consequently, there are many situations in which it is impossible to diagnose SO_2 damage by symptoms alone.

393. While many theories have been proposed to explain the mechanism of SO_2 injury, the actual mechanism is still in doubt. This is because the interconversion of sulphur compounds within the plant is still not fully understood. It is generally accepted, however, that the toxicity of the gas is primarily effected through its oxidation-reduction properties.

394. To this point in time, it has been difficult if not impossible to relate much of the controlled-environment fumigation research data to what actually goes on in the field. At best, it has provided

relative sensitivity values for various crops. Most of the HF and SO_2 monitoring data from pollution areas cannot be used to predict whether toxicity problems will arise. While the data provide relative atmospheric concentrations between locations and times, there is no way at present of converting absorption of HF or SO_2 per unit area per unit time into concentrations in the air which surrounds the plants. Even when concentration values are obtained, they in themselves are not predictive of injury unless chemical analysis and foliar symptoms data are available. Even then, the variability due to crop variety, cultural practices, growth conditions, environment and analysis can still make interpretation difficult much of the time.

395. It may be said that attempts to interpret the effects of gaseous pollutants, including fluoride compounds, on plants are complicated by the complexity of the factors affecting them. These include:

(a) concentration of the pollutant,

(b) length of exposure,

(c) amount of pollutant entering the leaves by means other than stomates,

(d) amount of intercellular space,

(e) cell wall thickness,

(f) translocation and assimilation or conversion of the pollutant,

(g) physiological condition of the plant,

(h) environmental conditions which control entry of and sensitivity to the pollutant.

396. Minor accumulations of zinc in the soil would probably be chelated, and thus rendered inactive. Excess zinc commonly produces iron chlorosis (bleaching) in plants, which can normally be remedied by application of limestone or phosphate which lower zinc solubility. Particulate zinc does not harm plants and will not affect the leaves directly. There is a possibility that an interaction will occur between sulphuric acid and zinc which may change the absorption patterns for the absorption of zinc by the plant. Information is needed on the zinc concentration in the soils and forages of the area before one can speculate on the possible significance of this element as a pollutant.

G. EVIDENCE FROM THE HEARINGS ON POLLUTION EFFECTS ON CROPS

397. Evidence was taken wherein damage was claimed to a wide range of plants, pasture, hay, corn silage, grain crops, vegetables, tomatoes, some flowers, trees and other ornamentals. In addition to claims of crop yield losses, claims were also made that markets for some crops were removed and/or in jeopardy.

398. This section deals with the alleged damage as reported in the testimony dealing with field crops: pasture, hay, corn silage, grain crops and other field crops. It is vitally important, in dealing with the evidence on such crop damage, that the information provided in Section B on "Soils as related to crop production" be noted and understood. It is most relevant.

399. The number of witnesses who testified is not necessarily an indication of the number of persons who reported or claimed damage of their field crops. The witnesses who were called to give evidence at the hearings on crop damage were representative of the area—an arrangement made by the Committee's Counsel and Counsel for the "Air Pollution Committee." However, every opportunity was afforded to any resident to give evidence if such was his or her desire. Excerpts from evidence are presented directly, in order that the type and extent of damage may be indicated. The witnesses are identified by name. The testimony given is to be found in Volumes VIII and IX.

(a) MR. J. CASINA, SR.

400. This man farms approximately 300 acres, 2½ miles E of Port Maitland, within the southern portion of the wind "cone" area.

Mr. Casina: *(p. 1370)*	*To talk about barley, I have had barley turn yellow and then the leaves turn white and drop off, just as white as that paper. That was in May of 1967.*
Mr. Brooks:	*What about your yields, how has that affected your yield?*
Mr. Casina:	*The yield in 1967 was—I think, around 30 bushels an acre.*
Mr. Brooks:	*And what were you looking for?*
Mr. Casina:	*Well, I have grown oats up to 90 and 100 bushels, and also in barley it was 90 to 100 bushels per acre.*

Mr. Brooks:	*And what did you get in 1967?*
Mr. Casina:	*Thirty—according to the adjuster from the Ontario Crop Insurance Commission — the 1966 yield I don't believe was as bad as in 1967.*

401. The following evidence speaks for itself:

Mr. Lamek: (p. 1393)	*Let us have a look at the extent of this 1967 damage, Mr. Casina, have a look at your field corn. I appreciate what you said a few minutes ago and I appreciate what Mr. Brooks said that this was a total claim but you are claiming 59 acres of field corn.*
Mr. Casina:	*That is right.*
Mr. Lamek:	*How many acres did you plant?*
Mr. Casina:	*59.*
Mr. Lamek: (p. 1394)	*I think you said you expected to get 50 or 60 bushels per acre on these 59 acres.*
Mr. Casina:	*Right.*
Mr. Lamek:	*And you are claiming what, 135 bushels to the acre?*
Mr. Casina:	*Right.*
Mr. Lamek:	*Mr. Casina?*
Mr. Casina:	*I don't know.*
Mr. Lamek:	*135 bushels to the acre, Mr. Casina, is that the damage that you say fluoride has done to your crop?*
Mr. Casina:	*I would say, yes.*
Mr. Lamek:	*Have you ever had 135 bushels to the acre? Your Counsel asked you and you said the best you had had was 85. Now what is it? Do you know what the best yield in the country was last year?*
Mr. Casina:	*I don't know.*
Mr. Lamek:	*Are you the best corn grower in the county, Mr. Casina?*

Mr. Casina:	*I consider myself a good grower, yes.*
Mr. Lamek:	*I think you estimate the value of those acres at 3 cents per lb. of corn?*
Mr. Casina:	*Three times 56* [lbs. per bushel], *that is $1.68.*
Mr. Lamek:	*Don't you think that is a little bit high for a bushel of corn?*
Mr. Casina:	*It could be at the present time.*
Mr. Lamek:	*What kind of price is it bringing this year?*
Mr. Casina:	*About $1.28.*
Mr. Lamek:	*You want $1.68 per bushel for 135 bushels per acre and this is the damage the fluoride emission is doing you?*
Mr. Casina:	*It could be.*

402. The same pattern followed when cross-examination took place in connection with Mr. Casina's tomato crop:

Mr. Lamek: *(p. 1396)*	*How many acres of tomatoes did you plant in 1967, Mr. Casina?*
Mr. Casina:	*Five.*
Mr. Lamek:	*And you are asking compensation for 57½ tons?*
Mr. Casina:	*Right.*
Mr. Lamek:	*How many?*
Mr. Casina:	*The number of tons is right in the report, you have got it there.*
Mr. Lamek:	*This 57½ tons and that is what you already sold to the canners?*
Mr. Casina:	*Yes.*
Mr. Lamek:	*Now you want to get paid for it?*
Mr. Casina:	*I think 11½ tons an acre I sold to the canners.*
Mr. Lamek:	*Now you are claiming 11½ tons an acre?*
Mr. Casina:	*That's right.*

Mr. Lamek:	*Do you think you can get 23 tons to the acre?*
Mr. Casina:	*I have got 30.*
Mr. Lamek:	*You told us before that your best tonnage in 1961 was 10 tons to the acre . . . ?*
Mr. Lamek:	*Will you agree with me for the four years 1957-60 your average tomato yield, according to your own figures, was 12½ tons per acre?*

403. Mr. Casina on several occasions complained that no individual from the Department of Agriculture and Food was concerned about his problem. Here is the evidence:

Mr. Gordon: (p. 1409)	*You have attended Vineland Agriculture and Experimental Station, either on your own or with other farmers, to discuss this air pollution problem?*
Mr. Casina:	*Yes.*
Mr. Gordon:	*Did you ever receive any correspondence from Vineland telling you anything about your crops?*
Mr. Casina:	*Never about crops, outside of suggesting a spray programme which would be carried out.*
Mr. Gordon:	*You have never heard anything at all? No letters, any correspondence?*
Mr. Casina:	*From Vineland?*
Mr. Gordon: (p. 1410)	*From Vineland or from anybody at Vineland?*
Mr. Casina:	*In relation to the damage that has occurred?*
Mr. Gordon:	*Yes.*
Mr. Casina:	*No.*

404. Mr. Casina seems to have a poor memory.

405. There are more than 60 entries, between January 12th, 1962, and November 1967, in the file of the Department of Agriculture and Food, including reports, copies of letters, and memoranda dealing with the problems of Mr. Joseph Casina, Sr. Of these, five were copies of letters written to Mr. Casina by Department of Agriculture and Food personnel stationed at Vineland. Included in the

file is, as well, a copy of a letter from Mr. Drowley to Mr. Fox (Vineland), a copy of which was forwarded to Mr. Casina.

406. It was essential that Mr. Casina should be further cross-examined about his statements and his repeated inference that no one was attempting to give him assistance. It should be mentioned here that, in addition to the attention being given to Mr. Casina and his problems as indicated above, there were, according to his own written and spoken words (p. 1351), many others who were helping him, as indicated below.

407. Mr. Casina stated that many persons interested in his problems were trying to assist. "A considerable amount of tomato plants were taken by Dr. A. N. L. Butler to make tests on," and (page 1352) "in 1962, I started corresponding and meeting many of the following specialists in their fields: Dr. A. N. L. Butler, W. B. Fox, G. A. Skinner, Mr. Gerald Weaver, Prof. C. B. Kelly, I. D. W. Smith, Hon. J. Allan, M.P.P., Deputy Minister of Agriculture Everett H. Biggs, W. B. Drowley, G. F. Gould, Mr. Thomas, Mr. Rayner, Mr. E. Wilcox, Norman Watson, M. Dreisinger, also personnel from industry."

408. He also stated that "test candles were set up on my property by Mr. G. F. Gould, Air Pollution Control Branch" (p. 1352).

Mr. Casina: *I brought it to the attention of Mr. G. F. Gould who took pictures of it. Samples of shrubs were sent to O.A.C. and Harrow Experimental Farm. I contacted Director W. K. Walker and Dr. A. N. L. Butler then approached processor and promised . . .*
"December 18, 1963—held a meeting at my farm with W. B. Drowley, Mr. W. B. Fox, Mr. W. I. Walker and Mr. W. Warnick and so it goes."

409. Further cross-examination revealed the following:

Mr. Gordon: *So, in effect, you are indicating that you didn't receive much advice or guidance from anyone that you contacted, so far as your crops were concerned.*

Mr. Casina: *No.*

Mr. Gordon: *Well, did you ever receive a letter in 1962 sent to you by Mr. Butler* [Vineland] *after he attended at your farm to take back some strawberry plants for analysis?*

Mr. Casina:	*Yes.*
Mr. Gordon:	*I thought you just said that you never heard from anyone at Vineland.*
Mr. Casina:	*Yes, I got correspondence from Vineland right here, not only from Vineland but from Butler and Fox* [Butler and Fox are both from Vineland.]
Mr. Gordon:	*Well, that was what I was asking you, whether there would be any other factors other than pollution which might have caused crop damage?*
Mr. Casina:	*Well, here is the letter here addressed to myself from Mr. Butler, Dr. L. Butler.*
Mr. Gordon:	*And it mentioned about thrips to your strawberry plants?*
Mr. Casina:	*Yes.*
Mr. Gordon: (p. 1411)	*And again, going back to 1962, did Mr. Butler, Mr. Wilcox and Mr. Smith attend at your farm to look over your crops?*
Mr. Casina: (p. 1412)	*I believe they did, yes.*
Mr. Gordon:	*Were you advised at that time that certain production factors, including weeds and disease, had taken a toll? All right, fine, and did you also have early blight which it was indicated suggested a weakness in your disease control programme?*
Mr. Casina:	*I don't recall . . .*
Mr. Gordon: (p. 1413)	*But you admit that you have had these problems (insects, disease, weeds, excess heat) over the years?*
Mr. Casina:	*Oh, I eliminate those problems.*

410. It should be pointed out here that in 1966, the year Mr. Casina complained of his barley crop, the barley disease "barley-root rot" was fairly common throughout most of Ontario (see evidence of Professor Tanner in cross-examination).

411. Mr. Casina was further examined by Mr. Gordon. It continued in part:

Mr. Gordon: (p. 1414) — *Well, I notice one of your neighbours or friends, Mr. McAlonan, claims 50 bushels* [of corn] *to the acre and he puts in a price of $1.60. Did you ever discuss with him his yield or what he felt his yields were?*

Mr. Casina: — *No, not with him.*

Mr. Gordon: — *And your neighbour, Mr. Carruthers, claims 50 bushels at $1.35.*

Mr. Gordon: — *Yes.*

Mr. Gordon: — *And then in your case, you claim 135 bushels to the acre at $1.67 (1.68)?*

Mr. Casina: — *I sold my corn in 1966 for 3 cents a pound.*

Mr. Gordon: — *And then Mr. Wright claims 125 bushels to the acre at $2.40 per bushel and then, at the other extreme, Mr. Karl claims 45 bushels to the acre at $1.25.*

412.
Commissioner: — *I wonder if Mr. Casina would please answer the question. Would you answer Mr. Casina, if you ever had, to your knowledge, a loss of crop on any of your farms due to weed, disease, insects, flooding or drought? Would you please answer YES or NO?*

Mr. Casina: — *Yes, in 1945.*

Commissioner: — *But you have not had any loss on your farm due to any of these things since 1945?*

Mr. Casina: — *No.*

413. The Committee, of course, does not sit as a Court-in-law, but the witnesses are sworn under oath before evidence is given. We are convinced that there has never been a farmer on this continent who has farmed over 300 acres of land who can swear to such a record!

(b) MR. ROBERT PAISLEY

414. Mr. Robert Paisley operates a 230-acre farm 3 miles NE of Port Maitland, just at the northern boundary of the wind "cone"

area. His evidence relative to livestock will be reviewed in Chapter V. In 1966, Mr. Paisley had 28 acres planted to oats and he "noticed that my crops—the oat crops—was not up to what it was the year before, and it wasn't up to what it was in the rest of the county." His yield was about 18 bushels to the acre, whereas in 1965 his yield was about 50 bushels to the acres. None of the oat plants was tested for fluoride. In the summer of 1967, he "had a streak right through the field that was damaged. Well, they were oats you know, they just kind of got yellow and went down." His wheat crop in 1967 was not good. That a part of his farm was not affected was indicated when he said "our frontage is about ¾ of a mile and we have never noticed any pollution on that part."

415. Mr. Paisley was cross-examined by Mr. Lamek:

Mr. Lamek: (p. 1187)	*And I understand that you grow corn pretty successfully?*
Mr. Paisley:	*Yes.*
Mr. Lamek:	*You have won awards for growing corn?*
Mr. Paisley:	*Yes, I have.*
Mr. Lamek:	*How did you do last year with your corn?*
Mr. Paisley:	*I did good, it was the second highest in the county.*
Mr. Lamek:	*It was the second highest in the county, you had a good corn yield?*
Mr. Paisley:	*Yes, but I had fluoride in hay on both sides.*
Mr. Lamek:	*But your corn was fine, then? How was the oat crop generally in the area in 1966?*
Mr. Paisley:	*Well, it was pretty good for the average of the county, I think it was 40 or 50 bushels to the acre.*
Mr. Lamek:	*And you said you got what in 1966?*
Mr. Paisley:	*I had 28 acres with about 480 bushels.*

(c) MR. J. CARRUTHERS

416. Mr. J. Carruthers farms 130 acres 2½ miles NE of Port Maitland, along the northern boundary of the wind "cone" area.

The evidence given by Mr. Carruthers in connection with livestock damage is recorded in Chapter V. He grew oats in 1967—"the

first time I have grown it in two years." The oats were grown on 13 acres of land drained by means of open ditches. His crop yielded a total of 150 bushels or 12 bushels per acre.

Mr. Gordon: (p. 1209) — *You indicated that you grew some oats and had a particularly bad yield. What would you expect, what would you expect to derive in terms of bushels per acre from your oats?*

Mr. Carruthers: — *Between 60 and 70.*

Mr. Gordon: — *Have you grown and achieved that in the past?*

Mr. Carruthers: — *In 1961 and 1962.*

Mr. Gordon: — *What other crops did you grow besides oats?*

Mr. Carruthers: — *Corn.*

Mr. Gordon: — *What happened there?*

Mr. Carruthers: — *I got about a 52 bushel acre average* [1966].

Mr. Gordon: — *How many acres of corn did you grow last year, 1967?*

Mr. Carruthers: — *60 acres.*

Mr. Gordon: — *What was your yield on that?*

Mr. Carruthers: — *About a 50 bushel average.*

Mr. Gordon: — *What would you expect normally?*

Mr. Carruthers: — *According to the fertilizer you put on, you can count on 100 bushels per acre.*

Mr. Gordon: — *Could you tell us what your lowest yield in the years were?*

Mr. Carruthers: — *I have reaped 70 to 75 bushels.*

Mr. Gordon: (p. 1211) — *Tell us what happened to your corn?*

Mr. Carruthers: — *When the crop got up about 10″ high, you cultivate it today and you go back in about two days and you think somebody went through it with a fire and singed all the ends of the leaves. This was not nitrogen which they tell us it is. If this starts in the leaf, it keeps going right up and that sets it back*

	for about 3 weeks and as a result in the fall of the year you don't get it off or it doesn't get ripe and the frost will cut your production down about 50 per cent.
Mr. Gordon:	*How had the yield compared—let us say, 1965, 1966 and 1967?*
Mr. Carruthers:	*I don't think there was over five bushels difference.*
Mr. Gordon:	*What is the highest yield that you have ever obtained from your farm in any given time or any given season?*
Mr. Carruthers:	*In 1961, 1962, I grew wheat and I got 30 bushels to the acre. The next two years I grew wheat and I could not get 18 bushels.*

417. Mr. Carruthers took off a hay crop in 1965 which was compensated for in 1966. "I just cut it and let it lay and the arbitrator wouldn't compensate me for it. In 1967, I cut it and let it lay and I don't expect to be awarded for it this year." (It should be noted here that Mr. Carruthers got rid of his cattle in 1966.)

418. Mr. Carruthers was cross-examined by Mr. Lamek:

Mr. Lamek: *(p. 1219)*	*Has the situation been any better this year as far as your crop damage goes?*
Mr. Carruthers:	*I can't see any improvement.*
Mr. Lamek:	*Do you think it is any worse?*
Mr. Carruthers:	*Well, let's look at it this way, up to a certain stage in the spring it might have been an improvement, it seems that if I have to get a crop planted and get it started, something happens to it.*
Mr. Lamek:	*Well, has it been worse this year, do you think, than in previous years?*
Mr. Carruthers:	*I think we got as much* [pollution] *in 1967 as we got in 1965.*
Mr. Lamek: *(p. 1221)*	*Did you hear Mr. Paisley give his evidence this morning?*
Mr. Carruthers:	*Yes.*
Mr. Lamek:	*About his corn crop last year?*

Mr. Carruthers: *Yes.*

Mr. Lamek: *How far are you away from his property?*

Mr. Carruthers: *A block and a half.*

Mr. Lamek: *And you heard him say he had a pretty good crop?*

Mr. Carruthers: *Yes.*

Mr. Lamek: *Your corn crop was somewhat not up to your expectations?*

Mr. Carruthers: *Right.*

(d) MR. H. RITTENHOUSE

419. Mr. H. Rittenhouse farms 110 acres 3½ miles E of Port Maitland, along the southern portion of the wind "cone" area. Evidence in connection with Mr. Rittenhouse's livestock damage will be presented in Chapter V.

Mr. Gordon: (p. 1226) *And what type of crops do you grow on this farm?*

Mr. Rittenhouse: *Oh, ordinary grain crops, even the dairy business.*

Mr. Gordon: (p. 1227) *Well, this hearing is concerned about allegations concerning pollution. Do you feel that your farm has been affected in any way and if so, would you tell us?*

Mr. Rittenhouse: *Well, I don't know, I haven't seen anything around our place. We feed the crops we grow and fortunately we have never had any trouble with our cattle going lame or loss of production or anything of that kind and I think according to what I hear around here we have been very fortunate. I have no explanation for it; I don't know why or anything about it.*

Mr. Gordon: (p. 1228) *Could you tell us anything whatever about the crops that you grow and what your experience has been with them, or is that the same?*

Mr. Rittenhouse:	*Well, we have had no difference in any crops. They may be loaded with this here trash, whatever it is, but as far as the growth is concerned I have never seen anything wrong at all.*
Mr. Lamek: (p. 1229)	*I see.*
Mr. Rittenhouse:	*We fill our silos off the acreage year after year, we have a good crop of hay, around half to three-quarters of a ton per acre and I am satisfied with the crops.*
Mr. Gordon: (p. 1234)	*And you haven't had any real complaints?*
Mr. Rittenhouse:	*No, I think we have been mightly fortunate. I am not saying about these other fellows. I don't understand it but those are the facts of the case. There are many people in this room today who heard me make these remarks before, that we have never had any trouble with our calves.*
Mr. Lamek: (p. 1235)	*Do you grow corn on your farm, Mr. Rittenhouse?*
Mr. Rittenhouse:	*Yes.*
Mr. Lamek:	*What kind of yield do you get to the acre?*
Mr. Rittenhouse:	*Well, we are not in that part of the business, we grow it mostly for silage.*
Mr. Lamek:	*But can't you give me an idea how much you raise to the acre?*
Mr. Rittenhouse:	*No, I would not know.*
Mr. Lamek:	*In any event, you are not unhappy with the amount you are getting?*
Mr. Rittenhouse:	*No, as I said before, I see no difference in our crop.*

(e) MR. E. HARVEY

420. Mr. E. Harvey farms with a brother, 200 acres, 2½ miles E of Port Maitland, just beyond the southern boundary of the wind

"cone" area. Evidence given by Mr. Harvey relative to livestock damage is presented in Chapter V.

Mr. Gordon: *(p. 1238)*	*What crops do you grow on your farm?*
Mr. Harvey:	*Hay and pasture and corn, cereal crops and sometimes wheat, oats and barley.*
Mr. Gordon:	*Can you tell us anything about what effect there has been on your farming operation in the last six years?*
Mr. Harvey:	*As far as corn goes, we haven't seen any bad results at all. Our corn has been good as far as growth and yield.*
Mr. Gordon:	*The growth and yield have been good?*
Mr. Harvey:	*Yes, but I don't know how much contamination is in the food.*
Mr. Gordon:	*Was last year a good year for corn, as far as you were concerned?*
Mr. Harvey:	*Yes.*
Mr. Gordon:	*And was your crop return on corn better last year than what it was in 1966?*
Mr. Harvey:	*Possibly it was because the fall of 1965 was wet and we did not get some of our ground ploughed until '66 and it did not do quite as well as it would have, but under the conditions it was a good crop in '66 but not as good as '67.*
Mr. Gordon:	*So you had no complaint insofar as your crop is concerned?*
Mr. Harvey:	*That is right.*
Mr. Gordon:	*What about the other crops that you grow?*
Mr. Harvey:	*We had a field of barley and I never noticed anything in the cereal grains, our wheat was good this year, better than I expected it would be, but we only had about 8 acres. We had a field of barley alongside the house and I suspect there was something the matter, it was planted late, in late May and it was as*

good a crop of spring grain as we have ever had, but just before it headed out, it turned yellow and actually just looked like the picture that Dr. Tanner had of the barley, but I was talking to Howard Henry, and I was explaining it to him and he convinced me that it was [not] *good barley all over the area and not just in our area. He said it had grown too fast and it was just the general conditions so I don't know.*

Mr. Gordon: *And so, insofar as the barley was concerned, it appeared to be all right with the exception of this condition that someone else convinced you was something other than fluoride.*

Mr. Harvey: *When it started to head out, there would just be an odd head come out and it just stood there for a while and it wasn't that dry, I mean, it was a bit dry but there was lots of ground moisture and then we got a small rain and it came out partially, but when it headed out there was only about ten kernels to the head. In fact, it was* [a type of] *barley but the peculiar thing about it is there was about half an acre in the same field sowed with oats and barley and it didn't seem to affect it. It was a six row barley. I believe it was Rodney oats, but I am not sure of the barley, but it was six row and they were good and I don't know the reason.*

421. Concerning a 25 acre field situated along the feeder canal, Mr. Harvey said: "We only thought we got more contamination on the back of the farm." Mr. Best examined part of this field and felt, according to Mr. Harvey, that "the alfalfa seemed to be spotted as though it was burned."

Mr. Gordon: *Do you know of any damage to any of your crops that would be directly attributable to any emissions from the plants in the area?*

Mr. Harvey: *Not definitely, we didn't put in a claim for the barley because my brother and I always felt that if the industry does the damage, they should pay for it, but on the other hand,*

	if it is something like poor management on our part, or disease, or anything else like that, we should take the loss.
Mr. Gordon:	*Have you had this condition in your barley before?*
Mr. Harvey:	*Well, we haven't grown straight barley.*
Mr. Gordon:	*Have you ever had conditions in your crops before that you might attribute to poor management or to disease or something like that?*
Mr. Harvey:	*Well, there is possibly conditions sometimes of poor management. There is always something like that.*

422. At this point in the cross-examination, Professor Tanner was asked by the Chairman to comment on the condition of the barley crop in Ontario in 1967.

Prof. Tanner: *(p. 1245)*	*Last year, in much of Ontario, or many parts of Ontario, the barley crop was one of the poorest barley crops we have had for some time; there was a fair amount of the diseased barley, yellow dwarf, also prevalent in Ontario, although I am not sure whether this area was as badly affected with the barley yellow dwarf. The fact that you had more in your pure stand than you had mixed with oats might make it seem as if it was barley yellow dwarf, which would not run as rampant in a mixed stand as it would in a pure stand. This is just speculation.*

(f) MR. LYALL NIECE

423. Mr. Lyall Niece and a brother farm 140 acres, 1½ miles SE of Port Maitland within the southernmost portion of the wind "cone" area.

Mr. Gordon: *(p. 1247)*	*And what type of crops do you grow?*
Mr. Niece:	*Mixed, wheat, oats and barley.*
Mr. Gordon:	*And could you tell us what crops you have grown, say, during the last five years, and what your experience has been with these crops?*

Mr. Niece:	*Always good up to last summer, we had one failure of oats.*
Mr. Gordon:	*So your crops were always good up until last summer, and it was only your oat crop that failed?*
Mr. Niece:	*One field.*
Mr. Gordon:	*Just tell us how many acres comprised that field and what you observed about it and what your yield was.*
Mr. Niece:	*Well, it was the first field we sowed and that was on the late side due to the weather, the 24th of May. I don't know if it was right on the 24th of May, but it was in that period and it came up nice and green about two inches and from then on it turned yellow and brown, something like that picture that was shown the last sitting we had here in February.*
Mr. Gordon:	*And your oats looked something like that?*
Mr. Niece:	*And the ten acres right beside it was sowed with corn on the 10th of June, which is late for corn, and it came up dandy.*
Mr. Gordon: (*p. 1249*)	*Now, what other crops other than oats have you grown on the farm?*
Mr. Niece:	*Wheat.*
Mr. Gordon:	*Wheat, any trouble with your wheat in the last five years?*
Mr. Niece:	*No.*
Mr. Gordon:	*It has grown normally and has harvested well?*
Mr. Niece:	*Yes, that is right.*
Mr. Gordon:	*Well, what other crops, if any, do you grow on your farm?*
Mr. Niece:	*That's it, wheat and oats and a hay crop.*
Mr. Gordon:	*And so from your experience, the only problem you have had was with the oats last year?*
Mr. Niece:	*That's right.*

Mr. Lamek: (p. 1251) *Mr. Niece, am I right in saying that the only damage to crops for which you have ever made a claim is with respect to this six or seven acres of oats?*

Mr. Niece: *That is right.*

Mr. Lamek: *How many acres of oats did you plant altogether last year?*

Mr. Niece: *Seventeen.*

Mr. Lamek: *Were the other ten acres all right?*

Mr. Niece: *Yes, that is right.*

Mr. Brooks: (p. 1251) *Were you selling any hay crops, Mr. Niece?*

Mr. Niece: *We always have, we have more hay and straw than we need for our own use and we try to sell it.*

Mr. Brooks: *Have you had any turned down?*

Mr. Niece: *Yes.*

Mr. Brooks: *When and why?*

Mr. Niece: *Along in the early part of January (1968) a gentleman drove in one day looking for hay and we made a deal with him and he said he would leave it for ten days or two weeks because he was getting a truck of his own and he needed a little time and all he was more or less doing that day was locating the hay. So we said, yes. Then on January 26th, 1968, we got a letter that said: "Dear Mr. Niece: We have heard through reliable sources that all your hay in your area is being burned due to fluoride poisoning; we are sorry that we cannot take your hay. Yours truly, Paul Hisen."*

Mr. Brooks: *Had your hay, in fact, been tested?*

Mr. Niece: *No, after this gentleman found out what they were doing with the hay up around our area, he didn't want to take it.*

Mr. Brooks: *This was without actual knowledge as to what the conditions were as far as your hay was concerned?*

Mr. Niece: *That is correct.*

(g) MR. PETER SHELESTOWSKY

424. Mr. Peter Shelestowsky farms 100 acres 2½ miles E of Port Maitland, near the centre of the southern portion of the wind "cone" area. This land is owned by ERCO. He worked the farm as a full-time farmer until 1967; in that year he worked part time for a construction contractor "because I now do not have any cattle."

Mr. Gordon: *(p. 1259)*	*What variety of oats would you grow?*
Mr. Shelestowsky:	*Rodney.*
Mr. Gordon:	*When would you plant the oats?*
Mr. Shelestowsky:	*Well, that depends on the weather, you know. Sometimes the 2nd or 3rd week in April, sometimes into May because it is wet and we can't plant.*
Mr. Gordon:	*What has your experience been with your oats, do they grow all right?*
Mr. Shelestowsky:	*No sir, not lately. I have six or seven years ago, I can't prove that I have 100 bushels per acre, but last year I had 30 bushels per acre. This is on the same land that I grew before with the tomatoes.*
Mr. Gordon:	*What did you notice about the oat plant, as such? Did you notice anything strange or unusual about them?*
Mr. Shelestowsky:	*That year, right in May, they turned yellow and stayed yellow all summer.*
Mr. Gordon:	*Did they grow to normal height?*
Mr. Shelestowsky:	*No, they grew that high* [witness indicates approximately one foot].
Mr. Gordon: *(p. 1260)*	*What about your wheat?*
Mr. Shelestowsky:	*I would say I have around 30 bushels now from wheat, 30 per acre.*
Mr. Gordon:	*Is that a good average?*
Mr. Shelestowsky:	*No, I have had better before.*

Mr. Gordon:	*When you say before it was better, how many bushels to the acre did you have?*
Mr. Shelestowsky:	*I have had 58 bushels per acre, that is the highest I had from wheat.*
Mr. Gordon:	*Did you notice anything unusual about your wheat plant itself? Was there anything wrong with the stems or the heads?*
Mr. Shelestowsky:	*No, I did not see anything.*
Mr. Gordon:	*Did you have either the wheat or the oats tested by anyone?*
Mr. Shelestowsky:	*No.*
Mr. Gordon:	*Was last year a good year for wheat, do you know?*
Mr. Shelestowsky:	*I had 30 bushels.*
Mr. Gordon:	*Yes, but did you consider that a good year for you, having regard to the climate and other factors?*
Mr. Shelestowsky:	*Somebody had it a little better probably, but that was all right.*
Mr. Gordon:	*All right, now you say that you grew some corn, how many acres of corn would you grow?*
Mr. Shelestowsky:	*I have 12 acres this year.*
Mr. Gordon:	*And how did the corn turn out?*
Mr. Shelestowsky:	*Oh, I had about 50 bushels.*
Mr. Gordon:	*Is that good so far as you are concerned.*
Mr. Shelestowsky:	*I don't know because I can tell you I never grew before, so I don't know how big the corn on my farm. I never grow before corn.*
Mr. Gordon: *(p. 1262)*	*And did you notice anything abnormal about the corn plants as they were growing?*
Mr. Shelestowsky:	*No.*
Mr. Gordon:	*They looked all right to you?*
Mr. Shelestowsky:	*They looked to me all right.*

(h) MR. RUSSELL ELKOW

425. Mr. Russell Elkow and three brothers farm about 500 acres 2½ miles NE of Port Maitland in the northern part of the wind "cone" area. Evidence concerning Mr. Elkow's cattle damage will be considered in Chapter V. Previously in dairy cattle, the Elkow brothers switched to beef cattle in 1967. During the past three or four years, they grew oats, barley, wheat, contract tomatoes, and corn. In 1967, they grew approximately 50 acres of oats.

Mr. Gordon: *(p. 1314)*	*What was your experience with that crop* [oats]*?*
Mr. Elkow:	*It wasn't bad, it was a pretty good crop.*
Mr. Gordon:	*Did you have any blight or any damage?*
Mr. Elkow:	*We did have some in July.*
Mr. Gordon:	*Did you know what caused it?*
Mr. Elkow:	*No, I didn't.*
Mr. Gordon:	*What did it look like?*
Mr. Elkow:	*It looked like rust, but I wasn't sure whether it was rust or not. These plants, when you grabbed hold of the plant and pulled it out of the ground there was no root connection.*
Mr. Gordon:	*What type of oats was this?*
Mr. Elkow:	*It was Garry oats, and the Rodney did not have this.*
Mr. Gordon:	*Well, your problem with your oats, did you relate it to any specific thing?*
Mr. Elkow:	*I don't know, I asked a few guys outside of the area and they told me they had the same thing. I was talking to some guy in South Cayuga and they have it.*
Commissioner: *(p. 1318)*	*I am told that root rot was very common in barley and oats in 1967, right across the province, Mr. Elkow.*
Mr. Elkow:	*I figured that, yes.*
Mr. Gordon:	*What about your barley, how many acres of barley did you grow last year?*
Mr. Elkow:	*Sixty.*

Mr. Gordon: *How did that crop turn out?*

Mr. Elkow: *Well, I think for the lateness of the season that we have a pretty good crop, 30 bushels.*

Mr. Gordon: *That is 30 bushels to the acre, did you have any complaints about the way it grew or the way the heads filled out?*

Mr. Elkow: *Well, July was awfully dry. I talked about this to one of the top barley growers in the area, and they had the same problem.*

Mr. Gordon: *What about your wheat, how many acres did you grow last year?*

Mr. Elkow: *We grew 28 acres.*

Mr. Gordon: *How did that crop turn out?*

Mr. Elkow: *One field was good and then the other two fields had turned, in the early part of the spring, a rusty colour. I had heard about that, this was a problem in Ontario . . .*

Mr. Elkow: *Yes, it was some kind of mosaic.*

Mr. Gordon: *How did the wheat harvest, did you get a good yield?*

Mr. Elkow: *Well, this one field had 30 bushels and it was put in late. The other field was in summer fallow and this is where we had the problem with the mosaic.*

Mr. Gordon: *And your corn, how many acres of corn did you grow last year?*

Mr. Elkow: *We had 200 acres.*

Mr. Gordon: *How did the corn turn out?*

Mr. Elkow: *Good.*

Mr. Gordon: *How many bushels did you get to the acre?*

Mr. Elkow: *We got the figures for the bushels and it ran around 26 per cent moisture and I think we figured it out to about 110 bushels. Now this is with the moisture, we never did get the dry figures.*

Mr. Gordon: (*p. 1317*)	*Did you figure that was a pretty good yield? Are you satisfied with it?*
Mr. Elkow:	*Well, for the lateness of the spring I was satisfied yes, I think the last field we got in was June 15th and this is late.*

(i) MR. TED McALONAN

426. Mr. Ted McAlonan farms about 70 acres 2 miles NE of Port Maitland, within the northern portion of the wind "cone" area. Testimony given by Mr. McAlonan in connection with livestock damage will be considered in Chapter V. Although Mr. McAlonan has a beef cattle and calf portion, he does "try to raise enough grain for my own use and possibly a couple of cash crops."

Mr. Brooks: (*p. 711*)	*What about the quality of this? Of the feed that you were taking off your own property, had it improved?*
Mr. McAlonan:	*Well, my hay has always been affected since 1965. Every time my hay has been tested, it has shown excessive amounts of fluoride.*
Mr. Brooks: (*p. 712*)	*Has any hay been removed from the barns or fields?*
Mr. McAlonan:	*Approximately thirty tons has been burned. I think it was the 12th or 13th of February, 1968.*
Mr. Brooks:	*Were you advised by anyone of the results or the reasons for that? Were the tests indicated?*
Mr. McAlonan:	*Well, it was on the high side, fifty parts per million, it was dangerous.*
Mr. Brooks:	*And your hay supply has now been replaced completely?*
Mr. McAlonan:	*Yes.*
Mr. Brooks: (*p. 713*)	*The first year of 1965 you received compensation; in your opinion and observations, had this situation improved at all?*
Mr. McAlonan:	*Well, I feel there has been an improvement since 1965, but I still think we are getting away too much of this* [pollution] *to eliminate the damage. Our damage as far as plant growth is concerned. I think it is just as bad in 1967 as it was in 1965.*

H. VEGETABLE CROPS, TREES, FLOWERS AND ORNAMENTALS

427. Once again, the Committee obtained evidence at the hearings which indicated that some damage to some types of vegetables occurred in certain farms or gardens and in other instances no such damage was noted on adjacent farms. Certain types of vegetables obviously are more susceptible to damage from the airborne pollutants than others. We will consider the evidence of each of the witnesses in turn:

(a) MR. RUSSELL ELKOW

428. Mr. Russell Elkow and three brothers farm about 500 acres, 2½ miles NE of Port Maitland within the northern part of the wind "cone" area.

Mr. Elkow: *We grew about a half acre of potatoes in our own*
(p. 1319) *garden.*

Mr. Brooks: *How did that work out in 1967?*

Mr. Elkow: *I have no complaints on the potatoes.*

Mr. Brooks: *And in 1966?*

Mr. Elkow: *I have no complaints. In 1965, we did have damage in our potatoes and this was on June 7th that I was talking about, and it singed the tops of the potatoes right off, but it never affected the yield.*

429. Mr. Elkow explained as well that they had damage to their evergreens in 1965, some in 1966 and some in June, 1967.

Mr. Brooks: *Did the evergreens recover, or have they recovered yet?*

Mr. Elkow: *They haven't got the rust on them now.*
(p. 1320)

Mr. Brooks: *What about others? Do they have maple trees?*

Mr. Elkow: *It doesn't affect the maples.*

Mr. Brooks: *What about the strawberries, what has been your experience with them in 1967?*

Mr. Elkow: *This is one crop we always did have damaged, 1965 was a bad year for strawberries.*

Mr. Brooks: *What about 1966?*

Mr. Elkow: *We had some damage and we had some damage in 1967.*

Mr. Brooks: (p. 1321) *Now, are there any other plants which you raised?*

Mr. Elkow: *Well, we have a row of rhubarb there, and it suffered in 1965 and 1966, and last year* [1967] *it was normal.*

Mr. Brooks: *Is there any other plant?*

Mr. Elkow: *Well, just the normal garden-like beans and sweet corn and things like that. There were cucumbers and beans, beets and carrots. These we grew for our own garden.*

Mr. Brooks: *Did you have any difficulty with those?*

Mr. Elkow: *Not as far as I know.*

(b) MR. ANTON KARL

430. Mr. Anton Karl operates a 63-acre market garden, 2½ miles NE of Port Maitland, within the northern portion of the wind "cone" area. Mr. Karl lives next door to the Elkow Brothers. He grows "tomatoes, strawberries, raspberries and all small stuff, even flowers; we used to raise raspberries. We are out of raspberries since 1964. We had problems since that time."

Mr. Brooks: (p. 1324) *Dealing with the year 1964, what was it?*

Mr. Karl: *Very poor quality* [peppers, raspberries].

Mr. Brooks: *What was it you first noticed about them?*

Mr. Karl: *Well, I went to Vineland that year with samples, to the experimental station, in 1964. We had problems with peppers and raspberries and with our fruit, just with small—I am referring to fruit trees.*

Mr. Brooks: *And what fruit trees are these?*

Mr. Karl: *Apples with mixed, but mostly apples and a few cherries. It is a family fruit, you know what I mean . . .*

Mr. Karl: *1964, that's right. I took pepper samples and from my raspberries. I knew that something was wrong*

with my raspberry patch, you know, the berries, the young shoots—see, your crop comes onto your young shoots and the young shoots were all singed.

Mr. Karl: *. . . because I am paying income tax every year except '65, I am not taxable because my crops were hurt badly. My income was nearly half in '65.*

Mr. Brooks: *Compared to '63?*

Mr. Karl: *Compared to '63 was down already. In 1962, I had my best crop ever, since I am farming.*

Mr. Karl explained that his strawberry crop had yielded:

in 1960—5,224 quarts; in 1961—4,939 quarts;
in 1962—6,853 quarts; in 1963—4,366 quarts;
in 1964—3,699 quarts; in 1965—1,146 quarts;
in 1966—2,100 quarts; in 1967—1,134 quarts.

431. In describing the yields recorded for his tomato crops, Mr. Karl said:

Mr. Karl: (p. 1327) *"And then I have my tomatoes here, this is field tomatoes contract per acre. In '60, per acre, 15¼ tons—this is per acre I am talking about. In '61, 16½ tons. In '62, 18¾ tons. In '63, it dwindled down to 10¾ tons. In '64, 10 tons. In '65, 8¼ tons. In '66 my crop yield went up again to 13 per acre. In '67, 13½ tons per acre, but my increased yield here was putting more population on our acreage* [more plants per acre]. *It used to be—I am a tomato grower for 20 years—we used to grow 2,700 plants per acre; so we increased this in the last three or four years with the new variety of tomatoes and we increased the yield, the tonnage; so really, 20 to 25 tons is possible and I think I would have that crop every year if nothing would interfere with my crop, honestly.*

432. It was evident that Mr. Karl, who in 1965 had difficulty in selling his berries on the market because of poor quality, attempted to obtain help from Vineland.

Mr. Brooks: *Mr. Karl, did you bring in any of the agricultural representatives, or have tests run on anything?*

Mr. Karl: *No, they would not stick their necks out. From 1964 and 1965, we went up to the experimental*

farm, not just me, but three or four of us, we were so panicked in our area when this occurred so severely. I would say in the middle of June, when we got singed so bad we could see it on the foliage and on the roads and all over, with the leaves all wrinkled up just like in the fall of the year, so we went up to Vineland again, so we explained up there our situation and we took samples down there and they promised us maybe next week they are going to come to see us, but they have not come yet.

Mr. Brooks: *That was in 1965?*

Mr. Karl: *Yes.*

Mr. Brooks: *And you have not been told even to date about this?*

Mr. Karl: *No, the Department of Agriculture and Food from Vineland came in with the Department of Health, and at our request.*

433. Mr. Karl testified, too, that his peppers had been badly affected, that of his eggplants "three-quarters of the leaves are burned . . . just like dry tobacco."

(c) MR. JOSEPH CASINA, SR.

434. Mr. Casina stated in his prepared brief (Volume IX) which he read at the hearings, that in 1961 he had had damage to one crop. "I could be at fault, but all my vegetable crops were below average." Of the 1962 crop year, he said, "my tonnage in tomato crops declined also my vegetable crops. In 1963 I was faced with a drop in crop production." He claimed, in evidence, "I believe from 1961 on it [yield of tomatoes] dropped from about 12 to about 5¾ tons per acre." He stated that he had, in 1961, purchased strawberry plants from Norfolk Farms and had planted an acre and a half to two acres and that during growth of the plant "the leaves, the edge of the leaves, they brown off and they curl up and dry up." He has not grown any strawberries since 1964. He claimed that other vegetables were affected as well and stated that the side of the plants and fruit trees "facing the industry" showed more damage than did the sides away from the industry.

435. Not only was some of Mr. Casina's evidence confusing, it was, as well, misleading. He admitted that, in 1965, an award for crop damage of some $6,100 had been made; that, in 1966, another

award of $4,700 had been made; and that he had made a claim for 1967 crop damage of $27,000. In response to questions on this point, the evidence is:

Mr. Casina: (p. 1328)	*My counsel has said that the letters of direction on the awards was changed. This is the reason for this. We were to put the full values and then let the arbitrators take off what you had and then put a value on it.*

436. Although Counsel objected to cross-examination of his witness, on the grounds that the 1967 claims (which had not been assessed) were made on a different basis than the 1965 and 1966 claims, nevertheless the Committee wished the examinations to continue. The Committee was concerned over some of the discrepancies in Mr. Casina's claims.

437.

Mr. Lamek: (p. 1386)	*Did you grow cucumbers in 1966?*
Mr. Casina:	*I think a few in the garden.*
Mr. Lamek:	*Did you claim for any damage to cucumbers in 1966?*
Mr. Casina:	*No.*
Mr. Lamek:	*Do you have a copy of that claim sheet in front of you?*
Mr. Casina:	*Yes.*
Mr. Lamek:	*Do you see the top line?*
Mr. Casina:	*Yes.*
Mr. Lamek:	*One: corn, sweet, there is a check mark under "check if applicable." On the right-hand side something, half acre, 1½ acres, cucumbers, melons; do you agree with that?*
Mr. Casina: (p. 1387)	*That was included in gardening that we had.*
Mr. Lamek:	*Do you agree there was a claim in respect of cucumbers in 1966?*
Mr. Casina:	*That's right.*
Mr. Lamek: (p. 1387)	*All right, did you grow melons in 1967, Mr. Casina?*

Mr. Casina: *That's right.*

Mr. Lamek: *Were they all right?*

Mr. Casina: *No, they had the leaf burn.*

Mr. Lamek: *Have you claimed for them?*

Mr. Casina: *I believe I have.*

Mr. Lamek: *Is this a copy of your claim sheet for the year '67, Mr. Casina?*

Mr. Casina: *That's right.*

Mr. Lamek: *Well, we find melons there?*

Mr. Casina: *That is included in the whole acre.*

Mr. Lamek: *Melons are eggplants, are they?*

Mr. Casina: *Melons, where did you see melons?*

Mr. Lamek: *I am asking you if you claimed melons in '67?*

Mr. Casina: *No.*

Mr. Lamek: *Now, Mr. Casina, I think I asked you if you grew melons in 1966?*

Mr. Casina: *I had a few in the garden, yes.*

Mr. Lamek: *Did you claim for them?*

Mr. Casina: *No.*

Mr. Lamek: *Let me show you a 1966 claim form again, do you have it there, Mr. Casina?* [Page 19.]

Mr. Casina: *19 what?*

Mr. Lamek: *1966 crop year. Here we go, I think this is the 1966 one, isn't it? I show you the top line, cucumbers and melons?*

Mr. Casina: *Included in the garden, I must have.*

Mr. Lamek: *(p. 1391)* *Did you grow pumpkins in 1966?*

Mr. Casina: *Yes.*

Mr. Lamek: *Did you claim for those?*

Mr. Casina: *I grew them in the garden, but I didn't claim for them.*

Mr. Lamek: *Are they specifically mentioned on your claim form?*

Mr. Casina: *No.*

Mr. Lamek: *(p. 1392)* *I show you the third line down, Mr. Casina, on the right-hand side; pumpkins, are you claiming those by name?*

Mr. Casina: *I don't see it on mine here.*

Mr. Lamek: *Is that 1965?*

Mr. Casina: *No, 1966.*

Mr. Lamek: *I will show it to you here. It says pumpkins, there is a specific reference to pumpkins.*

Mr. Casina: *Yes.*

(d) MR. J. OSTIRC

438. Mr. J. Ostirc farms 75 acres about 9 miles NW of Port Maitland and W of Dunnville (well outside "cone" area). He grows mostly tomatoes and peppers and oats, and had "orders from Toronto to take my stuff to Toronto. It was for 300 baskets of tomatoes I was supposed to deliver to my customer. At the same time the pollution came and they phoned me up and said 'don't bring that stuff in, it is diseased and poisoned.' I was supposed to move everything from his store." (See also Chapter IX, "Air of Death.")

Mr. Gordon: *What do you mean by "pollution"? What happened? What was the sequence of events?*

Mr. Ostirc: *I think the buyer saw the television and newspaper or something, and I never did know myself.*

Mr. Gordon: *(p. 1194)* *Were they pretty good looking tomatoes? Did they look all right to you?*

Mr. Ostirc: *The looked 100 per cent. I picked the baskets then I sell them. You see, I've got tomatoes like everything. If you have some that is wrong with what you call blotches or marks on, well you leave them in the field.*

Mr. Gordon: *(p. 1198)* *Well, I think I get what you are trying to say, but let me put it this way. You are saying that you live west of Dunnville?*

Mr. Ostirc: *Yes.*

Mr. Gordon: *You are not in the area of these two industrial plants?*

Mr. Ostirc: *No.*

Mr. Gordon: *But because of where you live and the fact that your containers and your truck has the name of Dunnville on them, that when you market your products, people turn them out because it comes from a pollutant area?*

Mr. Ostirc: *That's correct.*

(e) MR. D. DICK

439. Mr. D. Dick farms 125 acres, 8 miles NW of Port Maitland, outside of "cone" area. The same type of experience was related by Mr. Dietrick Dick who lives on the Grand River, between Dunnville and Cayuga. He has an acre of greenhouses and, as well, grows 30 to 40 acres of oats. Mr. Dick's story may be summed up by his evidence:

Mr. Dick: *(p. 1202)* *We never had any problem during the summertime but later in the fall our sales went back very fast.*

Mr. Gordon: *Just tell us, then, what did occur?*

Mr. Dick: *We don't think, in fact we know, we didn't suffer any pollution. We had wonderful crops. We had better crops than usual, but we noticed when people saw our name or our address, they just went by, so therefore, we left a lot of stuff in the fields.*

Mr. Gordon: *(p. 1203)* *You say when people saw your name or your address, where would they see this?*

Mr. Dick: *On the markets.*

Mr. Gordon: *Which markets?*

Mr. Dick: *Welland, Ontario.*

(f) MR. PETER SHELESTOWSKY

440. Mr. Peter Shelestowsky farms 100 acres 2½ miles E of Port Maitland, within southern portion of "cone" area. He is engaged in mixed farming, but particularly in growing tomatoes "and a little bit of strawberry, three-quarters of an acre of strawberry."

Mr. Gordon: *(p. 1255)*	*What can you tell us about your tomatoes during the last few years?*
Mr. Shelestowsky:	*In the last few years, they grow pretty good. I have a good yield. I have some plants that may have 200 fruit on the one plant. Another one in the middle of July, they start burning and that is three years in a row, '65, '66 and '67.*
Mr. Gordon: *(p. 1256)*	*Were you able to harvest these tomatoes?*
Mr. Shelestowsky:	*Yes, sir, but they are really hard to pick up . . . because so many of them are rotten.*
Mr. Gordon:	*So many of them were rotten?*
Mr. Shelestowsky:	*Yes, you must throw so many down before you pick up and put good ones in the hamper. So we pick up ourselves and we still have good high yields.*
Mr. Gordon: *(p. 1257)*	*Did this condition carry on during the three years?*
Mr. Shelestowsky:	*It started in 1965, yes.*
Mr. Gordon:	*But each year you were able to take a crop off?*
Mr. Shelestowsky:	*Yes.*
Mr. Gordon:	*What percentage of your crop would be marketable?*
Mr. Shelestowsky:	*I would say a third of the crop I lost.*
Mr. Gordon:	*You lost about a third and the other two-thirds were all right?*
Mr. Shelestowsky:	*Yes.*
Mr. Gordon: *(p. 1262)*	*Now, what about your strawberries? How many strawberries would you grow?*
Mr. Shelestowsky:	*I grow about ¾ acres of strawberries. It just keeps us busy, you know.*
Mr. Gordon:	*Did you market these strawberries to some people in the area?*

Mr. Shelestowsky: *They brown, they not going to grow either; we plant for this summer, they absolutely not grow. Just one part there, one part there, one down there. The rest they just burnt. I called Toronto, you know—the Department of Health. He took all the time take some sample, but I never had result. But we have no strawberry.*

Mr. Gordon: *(p. 1263)* *Did someone come over to take samples of your strawberries?*

Mr. Shelestowsky: *Oh, around in May.*

Mr. Gordon: *May, 1967?*

Mr. Shelestowsky: *1967, 1966, 1695, every year I call, you know, and they come and take a sample and they must be holding results, but I never heard anything.*

Mr. Brooks: *(p. 1264)* *Do you have any fruit trees, Mr. Shelestowsky?*

Mr. Shelestowsky: *Yes, I maybe have about 15 apple trees.*

Mr. Brooks: *Do you maintain them, spray them regularly?*

Mr. Shelestowsky: *No.*

Mr. Brooks: *Have you had trouble with them, additional trouble or—*

Mr. Shelestowsky: *Well, they are not going to grow now, they are the same like tomatoes, all the leaves turn brown. You know . . .*

Mr. Brooks: *It happens to the maple trees, too, does it?*

Mr. Shelestowsky: *Yes, sir.*

Mr. Brooks: *What other types of trees do you have around your place?*

Mr. Shelestowsky: *Well, I have some spruce trees.*

Mr. Brooks: *Have they been affected?*

Mr. Shelestowsky: *Yes, the same thing there.*

(g) MR. A. HOTO

441. Mr. A. Hoto lives in the village of Stromness on one acre of land, 1 mile E of Port Maitland within the "cone" area. He

stated that in 1963 damage from industrial pollution became evident. His strawberries and raspberries were affected as well as the garden vegetables such as tomatoes and onions. He has, since 1964, abandoned gardening. Some spruce and cedar trees have been affected and his "white ash trees around [his] line fence and they are particularly vulnerable to pollution."

I. SUMMARY

442. On those farms where good crop management was practised and where the normal seasonal effects of moisture, drainage, insect and plant diseases were recognized and evaluated, the evidence shows that there was little decrease in yields or quality of shelled corn, winter wheat, oats or barley, in the so-called affected area, in comparison to the rest of the county (see Figure 7, which appears on page 262). In 1966, the barley yield was considerably lower than in the rest of the county. In 1966, the barley yields were considerably lower than in the preceding year and in the succeeding year, but it is known that the disease "barley yellow dwarf" was widespread throughout Ontario that year. However, the evidence did show that more than one field of cereal grain on more than one farm had been adversely affected, in a line pattern fashion, by some pollutant. Whether this was caused by a massive emission of SO_2 or of fluoride is not specifically known. It may be assumed that the damage was caused by fluoride.

443. There is no question that certain of the pasture crops, hay crops, and leaves of corn, have been "contaminated" in the sense that high concentrations of particulate fluoride have been deposited on or absorbed by leaves. The effects on livestock of consuming such fodder will be discussed in the next chapter.

444. On the other hand, there was no consistent evidence produced to show that the ripened seeds of the cereal grains, corn, wheat, barley, oats, etc., were abnormally high in fluoride. Experimental work recorded in the literature strongly supports this contention. The literature clearly shows, too, that different varieties of the same species, e.g., corn, show marked differences in their susceptibility to the fluoride ion.

445. The acreage of tomatoes grown in the area is considerable. The sale of tomatoes is of great economic importance to many of the farmers and market gardeners. Although evidence was presented which indicated that some damage to tomato crops by pollutants had occurred, other evidence denied any decrease in quality or in yield per acre. Under the system of estimating and assess-

ing damages and/or losses in the crop years 1965 and 1966, it is impossible to secure basic facts. Nevertheless, we accept evidence that in some instances damage to tomato crops has occurred. The greatest loss, however, was vastly more significant and far-reaching—it was a "stigma" loss, a loss of special markets and of near total income resulting from an unwarranted emotional fear that the products from "Dunnville" were inedible, contaminated and dangerous. Such attitudes are damaging and insupportable. The CBC in particular should recognize the guilt which bears heavily on its shoulders.

446. The evidence also substantiates the claims that many of the "area" market gardeners have suffered losses as a result of airborne pollutants. Recognizing that different species of vegetables and fruits show markedly different degrees of susceptibility to fluoride and to sulphur dioxide, it is questionable if the growing of some types of plants, e.g., pepper plants, is feasible in the area until the "air pollution" is controlled to a much more satisfactory level. Indeed, the future of market gardens, wherein "susceptible" types of vegetable, fruit and berry may be grown successfully, is in jeopardy until air pollution control is further improved.

447. At the same time, evidence does show that most vegetables, when variety susceptibility is also considered, can be grown successfully in areas where the concentration of fluoride and/or sulphur dioxide is considerably higher than is to be found ordinarily in Moulton and Sherbrooke Townships.

448. Damage to those portions of some evergreens and some deciduous trees on the windward side of the prevailing wind has been established. Whether such damage is the result of sulphur dioxide or fluorides has not been established, but it is suspected that most of it has resulted from high fluoride emissions into the air. The leeward sides of such affected trees have shown less or even no damage. The evidence also showed that a few trees and some shrubs, varieties unknown, have died. Whether this is a result of air pollution or of some other cause, is, of course, unknown. Evidence of specific damage, as indicated in various parts of this report, is excepted from the above statements.

CHAPTER V

Effects of Air Pollution on Farm Animals

A. INTRODUCTION

449. The International Encyclopaedia of Veterinary Medicine (1966) introduced the subject of fluorosis with these words:

> "Fluorine occurs throughout the world in soils, plants, water and animal tissues. Because of its high chemical reactivity, it is found in nature in a combined form. Animals normally ingest variable amounts of these fluorides throughout their lives. If amounts above the threshold or critical level are ingested over long periods of time, fluorine poisoning may result. This may be either acute or chronic. Fluorine is accumulative in the animal body so long as the animal continues to ingest a constant or increasing amount. The problem in livestock is usually with chronic toxicity."[47]

450. As far as we know, the earliest reported occurrences of fluorosis in animals followed periodic volcanic eruptions in Iceland as early as 1100 A.D. More detailed accounts have been presented by Røholm, based upon records following the eruption of Hekla in 1845. Many animals died of acute poisoning within a matter of weeks, but the majority began to show symptoms of a chronic toxicosis about a year later. The description of the signs and symptoms of a chronic toxicosis was essentially as we know them today. Such volcanic eruptions, with the resultant fluoride contamination were "Acts of God." The occurrences attributable to man, through in-

dustrial processing of fluoride-containing ores or rock, date back to the early manufacturing of aluminum (recorded in 1912), superphosphates (recorded as early as 1891), hydrofluoric acid (recorded in 1902), glazed bricks (recorded in 1913), processing of copper (reported in 1883), the manufacturing of glass and enamel (recorded in 1878, 1903, 1907) and more recently of high octane gasoline. The increase in general industrialization in the U.K. and Europe, and later in the U.S.A., saw a corresponding increase in the number of factories which emitted effluents of silicon fluoride or hydrogen fluoride or gave rise to dust containing various fluoride compounds. Throughout the relevant literature, there is no doubt in the respective authors' reports that the fluoride intoxication of livestock resulted primarily from the ingestion of forage crops which had been contaminated by the fluorides emitted from the various industrial plants.

B. NUTRITIONAL CONSIDERATIONS

451. The nutritional aspects of the fluoride problem in livestock production must be considered in a general way prior to the discussion of the several detailed phases of the subject. Professor S. J. Slinger of the Department of Nutrition, University of Guelph, a Consultant to the Committee, prepared a brief in this connection. Once again, as has been the practice of the Committee, parts of the brief have been used in the report without necessarily referring again to the source. (See also the reviews—National Academy of Sciences, N.R.C. (U.S.A.) Publication 824, (1960)[48]; Mitchell and Edman's Nutritional Abstracts and Reviews, 21, (1951-1952)[49]; and the review by Cass (1961)[50]—as used by Slinger.)

452. Fluoride is present in varying amounts in all feeding stuffs for livestock and can be continuously consumed below certain levels without harm to the animal or effecting economic loss. When ingested in amounts above the critical levels, fluoride is toxic and the degree of toxicity is dependent on the level of intake, the time period of excessive intake, the fluoride compound consumed, and the species of animal involved.

453. The hazard for livestock may stem from excessive fluoride in mineral supplements, in contaminated forages, in drinking water, in feed supplements, or combinations of the above. The association of fluoride with phosphatic minerals is of interest in both plant and animal nutrition. Fluoride contributes to the insolubility of phosphate deposits and is thought to be essential to their preservation through the ages.

454. Water supplies having the highest concentrations of fluoride are obtained from deep wells where the fluoride appears to originate, not from leachings from the surface but from deep-seated rock formations. Sea water contains from 1.0-1.4 p.p.m. of fluoride.

455. Of the feedstuffs used in livestock feeding, the cereals—corn, wheat, oats, barley, rye and wheat milling by-products—normally contain from less than 1 to up to 3 p.p.m.; soybeans contain from 1 to 4 p.p.m. Meat meal and fish meal protein supplements often contain considerable quantities of ground bone; bone in general, and particularly bone from marine sources, often contains fairly high levels of fluoride. Numerous calcium phosphate supplements are used routinely in formulas for animal feeds. Some of these will contain 4,000 to 15,000 p.p.m. or more of fluoride and can make a very significant contribution to the fluoride intake of animals through being mixed in the feed or fed as part of a mineral supplement. Some non-phosphate limestones mined in Ontario contain up to 2,000 p.p.m. of fluoride.

456. Forages grown in areas outside of zones known to be industrially contaminated contain 2-75 p.p.m. of fluoride, while those grown in areas where fluoride is emitted into the atmosphere in large amounts may have concentrations ranging from those found in normal forage up to 500-1000 p.p.m. In contrast to the forages, the cereal grains grown on industrially contaminated areas have fluoride concentrations similar to the normal levels for the various grains, generally less than 10 p.p.m. and with an average of 3-5 p.p.m. Where corn is stored in an open crib, which is a popular procedure, it would be expected that considerably higher levels could be achieved through particulate matter adhering to the grain surface.

457. The quantitative determination of fluoride in organic matter is not simple and the possibility for error is great. In addition, the taking of suitable samples for analysis requires considerable "know-how" and there seems little doubt that sampling errors are responsible for many of the apparent discrepancies in fluoride levels reported in the literature and in reports submitted at these hearings.

458. The solubility in water and weak acid has a considerable influence on the absorability of fluoride by animals. The following table shows the solubilities of a number of compounds:

TABLE XV

WATER SOLUBILITY OF SOME FLUORINE COMPOUNDS

Compound	Fluorine Content Per Cent	Solubility at 25° in water g/100 ml*	HCl 0.5% g/100 ml**
NaF	45.46	4.210	4.190
CaF_2	48.63	0.0017	0.032
Na_2SiF_6	60.57	0.759	0.942
K_2SiF_6	51.70	0.076	—
$BaSiF_6$	40.68	0.024	—
Na_3AlF_6 (Cryolite)	54.26	0.039	0.270
Na_3AlF_6 (Synthetic)	52.50	0.063	—

* From *Fluoride Intoxication* by Kaj Røholm, (1937)[11]

**From Mitchell and Edman (1951-1952)[49]

459. From the above table, it is seen that sodium fluoride (NaF) is by far the most soluble compound, and that calcium fluoride (CaF_2) is the least soluble; these differences are reflected in their relative toxicity. Fluoride in rock phosphate is reasonably insoluble, relative to NaF, and evidence indicates that the tolerance of animals for rock phosphate-fluoride is about twice that for the fluoride in sodium fluoride. The hydrofluoric acid (HF) ingested by cattle would be quickly and completely converted to NaF in the presence of sodium bicarbonate in the copious saliva; this is also true for many of the other fluorides which would be contained in the emissions from a phosphate plant. However, some of the emissions are particulate dusts, containing the relatively insoluble forms of fluoride. The analyses for fluoride conducted by the Ontario Water Resources Commission are for total fluoride, as are the levels indicated by the "lime candle" technique employed in the area under study. The lack of distinction between soluble and insoluble forms of fluoride in the analyses renders the analyses much less valuable than would otherwise be the case.

460. As Professor Slinger pointed out in his review, there are conditions wherein lower levels of fluoride intake may produce toxicosis. The physiological effects of fluoride are a function of time as well as of concentration. The long term ingestion of marginally

toxic levels of fluoride through periods of physiological stress such as inadequate nutrition, marked changes between seasons, growth, lactation, pregnancy and disease, may lead to toxicosis. The hazard of fluoride toxicosis caused by physiologic stress varies with the fluoride content of the feed. This is illustrated in the following two tables from Publication 824, National Academy of Sciences (1960)[48].

TABLE XVI

ESTIMATED FLUORINE INTAKE OF BEEF AND DAIRY CATTLE UNDER SPECIFIC CONDITIONS AND WITH SPECIFIC F LEVELS IN FEED

					F intake with indicated levels of F in feed					
	Body Weight lbs.	*(kg)*	*Daily Feed dry lbs.*	*(kg)*	*30 p.p.m. mg.*	*mg/kg*	*40 p.p.m. mg.*	*mg/kg*	*50 p.p.m. mg.*	*mg/kg*
					DAIRY CATTLE					
Growing	300	(136)	7.75	(3.52)	106	0.78	141	1.00	176	1.30
	600	(272)	13.50	(6.13)	184	0.68	245	0.90	303	1.10
Maintenance	900	(408)	13.50	(6.13)	184	0.45	245	0.60	303	0.74
	1200	(545)	16.20	(7.37)	220	0.40	294	0.54	368	0.68
	1500	(681)	19.80	(9.00)	270	9.40	360	0.53	450	0.66
Lactating	900	(408)	30.00	(13.60)	408	1.00	545	1.30	680	1.70
	1200	(545)	33.00	(15.00)	450	0.82	600	1.10	750	1.40
					BEEF CATTLE					
Growing	300	(136)	9.00	(4.10)	123	0.90	164	1.20	205	1.50
	600	(272)	14.00	(6.55)	196	0.72	262	1.90	326	1.20
Maintenance	1000	(454)	18.00	(8.20)	248	0.55	330	0.73	412	0.90
Lactating	1000	(454)	28.00	(12.70)	380	0.84	507	1.10	634	1.40

461. The data in the above table indicate that the fluoride intake for rapidly growing cattle is greater than that for cattle on a maintenance regime. Because of their high feed requirements, lactating dairy cows consume more fluoride per unit of body weight than rapidly growing stock, this being exacerbated with the smaller bodied animals. These same principles, according to Professor Slinger, apply to beef cattle, excepting that, because of their relatively more rapid rate of growth and lower milk production potential as compared with dairy breeds, the intake of fluoride is higher for rapid growth in milk-producing beef animals.

TABLE XVII

ESTIMATED EFFECT OF SEVERAL LEVELS OF MILK PRODUCTION, CERTAIN LEVELS OF DIETARY F, AND WEIGHT LOSS IN LACTATION, ON F INTAKE PER UNIT OF BODY WEIGHT — 1200-LB. DAIRY COW

			Effect of loss of wt. due to lactation on mg. F ingested per kg. of body wt.		
	DM/ day*	*F/ day*	*1200 lbs.*	*1100 lbs.*	*1000 lbs.*
	lbs.	*mg.*			
30 p.p.m. F					
Maintenance	15	202	0.37	0.40	0.45
40 lb.milk/day	33	450	0.83	9.90	1.00
60 lb.milk/day	38	515	0.95	1.04	1.15
80 lb.milk/day	43	585	1.08	1.17	1.30
40 p.p.m. F					
40 lb.milk/day	33	595	1.08	1.18	1.32
60 lb.milk/day	38	685	1.25	1.37	1.52
80 lb.milk/day	43	780	1.44	1.55	1.74
50 p.p.m. F					
40 lb.milk/day	33	745	1.37	1.48	1.65
60 lb.milk/day	38	855	1.56	1.71	1.90
80 lb.milk/day	43	975	1.78	1.94	2.16

* DM = dry matter

462. The data in the above table show that, as milk production increases, feed and therefore fluoride intake increases. Also, as body weight declines during lactation, the increase in fluoride intake per unit body weight increases. The combination of high level of milk production and declining body weight adds up to make levels of fluoride, which are not hazardous for maintenance of cattle, quite unsafe under these conditions,

463. In his evidence, (Vol. VII, p. 979) Dr. Suttie correctly pointed out that the figures in the above tables assume that all components of the diet contain the same level of F and, while the data are quite correct, they may not have direct application to a

pollution situation. Since the increase in feed intake, as dairy cows increase their milk production rate, is mainly in the concentrate portion of the diet, the high-producing cow may well not suffer unduly if the concentrate portion of the diet contains less than the toxic level of fluoride.

464. However, it may be well to note that the Feeds Regulations (1967) of the *Canada Feeds Act* permit 90 p.p.m. of F in complete balanced feed for cattle, which is in the toxic range even if all the F in the concentrate is in the less soluble forms. Where concentrate is being mixed with locally produced grain, as was the practice of a number of farmers in the Port Maitland area, one would expect some increase in F in the readily soluble forms. Furthermore, it is common practice to feed dairy cows supplementary mineral to balance that lost during the milking period (lactation). Mineral feeds for cattle may legally contain 2,000 or 3,000 p.p.m. of F, depending on their phosphorus content. At least one commercial concern, purveying mineral mixes to farmers, recommends mixing 2 per cent of their mineral in the feed for normal production and 4 per cent for high-milk-producing dairy cattle. The addition of a 4 per cent mineral mix, containing 3,000 p.p.m. of F, would add to the concentrate 120 p.p.m. of F which, without any other source in the concentrate, is well within the toxic range. Based on these considerations, it would appear unwise to assume that an increase in concentrate consumption by cattle will invariably dilute the levels of F provided by forage from a polluted area.

465. High-producing cows also drink considerably more water than low producers; this could accentuate the problem of the high producer, particularly where water contains considerably more than 1 p.p.m. of fluoride. Water-borne fluoride has been reported to be more readily available than an equivalent amount present in dry feed.

466. Assessment of difference in fluoride sensitivity of animal species is difficult unless the conditions under which they are assessed are standardized with respect to source of fluoride, comparable life-span period, and other criteria. Under conditions approximating these requirements, with raw rock phosphate as the fluoride source and growth inhibition as the criterion, data on species' tolerances were obtained at the Wisconsin Experimental Station. These data are shown in Table XVIII and are expressed as the maximum level of fluoride ingestion compatible with normal growth[48].

TABLE XVIII

LEVELS OF INGESTION OF FLUORIDE FROM RAW ROCK PHOSPHATE COMPATIBLE WITH NORMAL GROWTH OF VARIOUS SPECIES FROM WEANING TO MATURITY

Species	*Fluoride per kg. body wt. per day (mg.)*
Cattle (dairy cows)	2-3
Rabbits	11
Swine	8
Rats	8-10
Guinea Pigs	18
Poultry	35-70

467. Other evidence has shown that, of the farm animals, cattle and sheep are the most susceptible to fluorine toxicosis. Burns and Allcroft (1964)[51] list the order of susceptibility of farm animals as calves, dairy cows, beef cattle, sheep, horses, swine, and poultry. Fluorosis in swine has not been a major problem. Horses appear to be reasonably resistant to fluorine poisoning and authenticated cases of fluorosis in horses in the United States, at least, are rare. Poultry, of all farm animals, are probably the most resistant to fluoride and are generally considered to present no problem in so far as fluorosis is concerned.

468. According to Professor Slinger, there is reference in the literature that swine on pasture tolerate considerably higher levels of fluoride than those raised in dry lot. The mechanism for this alleviation has not been determined. Since legume pasture is high in calcium relative to phosphorus, it may be that the formation of the relatively insoluble CaF_2 is responsible, in part at least, for this alleviation.

469. Because of the alleviating effect of pasture on fluoride toxicity, one should be cautious in extrapolating experimental results based on dry lot feeding to animals kept on fluoride contaminated pastures.

C. RELATIVE HAZARD OF INDUSTRIAL FLUORIDE CONTAMINATION FOR DIFFERENT CLASSES OF LIVESTOCK

470. Poultry: Broiler chickens constitute the main meat bird raised in Ontario at the present time. Broilers are raised in confinement, with no access to pasture, and for the most part receive

complete commercially-prepared feeds. It requires about nine weeks for a bird to grow out to market weight and, even if the broilers were using a source of water contaminated with several p.p.m. of F, and if all the grain portion of their diet was grown in heavily contaminated soil and stored in a crib in the atmosphere of an industrial plant emitting fluorides, it would be very difficult to produce fluorosis in broilers.

471. Turkey broilers are in much the same position as chicken broilers, excepting that the time to produce a market bird is somewhat longer (12-15 weeks). It requires 20-24 weeks to produce a heavy roaster-type turkey. There was a time when most turkeys were raised on range, with free access to pasture and a free-choice supply of concentrate feed. Even under these circumstances, only about 10-15 per cent of the total feed of the turkey was obtained from pasture, with the remainder being purchased feed or home-grown feed, relatively low in fluoride.

472. As the turkey industry has evolved, there are fewer turkeys being raised on range, so that most turkeys today have no chance to eat contaminated forages. These facts, coupled with the relatively high tolerance of turkeys to fluoride, make it almost impossible for turkeys to be affected by this type of industrial pollution.

473. There also appears to be little chance that table egg- or hatching egg-producing birds would be affected. Such birds are almost invariably reared indoors for the first 10 weeks or thereabouts; some flocks are then placed on pasture for about a 10-week period, following which they are housed again until the end of the laying period. Egg-type birds are usually marketed at around 18 months to 2 years and meat-type birds at 14-18 months of age. Again, as in turkey production, there is a rapid trend to all-confinement rearing of laying and breeding stock. Chickens are relatively poor foraging animals and they will not usually consume more than about 15 per cent of their total feed intake as pasture.

474. Swine: More and more market hogs are being raised in confinement, with no access to pasture. They are usually fed commercial feeds but, even if they were to be fed a commercial supplement mixed with home-grown grain, their tolerance is such that no hazard would be involved, even with grain containing 10-20 or more p.p.m. of fluoride. Hogs are usually marketed at 6 months of age or less. Brood sows are sometimes pastured during the gestation period. The known alleviating effect of pasture on the development of fluorosis may well be a factor in preventing harmful effects

in some sows. In a report by Largent (1949)[53], in which he described the effect on farm animals in close proximity to a phosphate plant in the U.S.A., it is of interest that, while the cattle were affected and showed severe clinical signs of fluorosis, none of the farmers in the area reported similar difficulties with swine. It would appear that, because of their relatively high tolerance and their reliance on concentrate rather than roughage feeds, there is little danger of fluorosis in swine.

475. Horses: Largent also reported that, even under conditions where cattle were severely affected as a result of emissions from a phosphate plant, no difficulties were encountered in horses. He suggested that the greater susceptibility of the cow, as compared with the horse, may be that food eaten by the cow may remain in the gastro-intestinal tract for a longer period of time than would be required for its passage through the digestive tract of the horse. Thus, such relatively insoluble material as rock phosphate would have more opportunity to dissolve and have the F absorbed in the cow. Whatever the reasons, it is generally recognized that the horse is more tolerant to fluoride than cattle or sheep.

476. Sheep: Bosworth (1941)[54] has reported on the effects of emissions from a brickworks in England. While definite signs of fluorine intoxication were observed among cattle, they could obtain no evidence to suggest that other animal species were clinically affected. In one flock of breeding ewes, many animals showed the typical dental changes of fluorosis but otherwise appeared to be in quite good health, although they had been kept for a long time on pastures which were sufficiently contaminated to have caused obvious bone lesions in cattle.

477. Cattle: Since cattle are normally heavy forage consumers and eat relatively little grain, they are exposed, to a greater extent than other species, to industrial contamination with fluorides. Coupled with this is the fact that cattle can tolerate less fluoride than other species and have a longer life-span in which to consume fluoride. These factors make the hazard of industrial contamination greater for cattle than for other animals.

478. Because of the greater physiological stresses to which they are exposed, lactating dairy cows face a greater hazard from fluoride excess than other classes of dairy animals and all classes of beef cattle.

479. Suggested levels of fluoride, on the basis of present information, have been proposed by the National Research Council (U.S.A.) and are presented in Table XIX.

TABLE XIX

SAFE LEVELS OF FLUORINE IN THE TOTAL RATION OF LIVESTOCK

Species	*Sodium fluoride or other soluble fluoride*	*Phosphatic limestone or rock phosphate fluoride*
	p.p.m.	*p.p.m.*
Dairy Cow	30-50	60-100
Beef Cow	40-50	65-100
Sheep	70-100	100-200
Swine	70-100	100-200
Chickens	150-300	300-400
Turkeys	300-400	— —

480. Bees: (While not farm animals, bees are of importance to farmers and are considered at this time as a matter of convenience.) The effect of atmospheric fluorine on bees, according to the evidence presented by Professor Tanner, is not fully understood at this time. Several variables have led to some apparent contradictions in the literature. Murizio (1956)[55] reported on a field study in Switzerland where atmospheric fluoride pollution was shown to be causing heavy damage to bees in that particular area. His conclusion was that the poisoning was due to the constant presence of fluoride compounds on the flowers of various types of vegetation. The compounds were presumed to have been collected by the bees with the pollen and carried into the hives. Jackimowicz of France (1964)[56], according to Professor Tanner, reported that bees, taken from hives one to 20 kilometers from an aluminum plant producing 52,000 tons per year, contained fluorides but that the amount of fluoride in the bees was not related to the distance from the plant. He found that some bees contained as much as 10 times the amount of fluoride considered to be lethal. The authors suggest that bees living near a major sources of fluoride pollution may develop a resistance. They contend that, if fluoride poisoning were a real danger, bee keeping in the contaminated area would have died out—which it has not.

481. Another study, conducted by Toumanoff and Jackimowicz (1959)[57], showed that caged honey bees which were fed sugar solutions containing sodium fluoride, in concentrations as high as 200 p.p.m. (approximately 100 p.p.m. F), appeared to possess a marked resistance to fluoride. Again, using Tanner's evidence, according to Guilhoun of France (1962)[58], the amount of fluoride normally found

in bees is quite variable, depending upon foraging activity, nectar sources, wind direction, and topographical conditions. In so-called non-polluted regions, the average fluoride content is 1 microgram per bee. Several other studies apparently have shown 1 microgram to be a maximum figure. The "Ld 50" (lethal dose for 50 per cent of the population), according to Tanner's evidence, has been shown to be 5 to 10 micrograms for sodium fluoride, ammonium fluoride, hydrogen fluoride, and cryolite.

482. Guilhoun and his associates, reporting in 1962, showed that in rural, non-polluted areas the levels of fluoride in bees varied from 0.02 to 0.98 micrograms per bee—in other words, less than 1 microgram per bee. In low-pollution areas, such as are found in large towns or industrial areas, the fluoride content was found to vary from 1.5 to 10 micrograms per bee. In the area of an aluminum plant (where fluoride emissions are generally found) the following data were obtained:

TABLE XX

FLUORIDE CONTENT IN BEES NEAR ALUMINUM PLANT

Distance from Plant	*Fluoride content, Micrograms per bee*
5 kilometers	6-13
600 meters	9-30
600 meters, resting bees	9-10
600 meters, actively foraging	18-30

483. These data show that there is a marked difference in fluoride content, depending upon whether or not the bee is actively foraging for nectar, etc. Guilhoun's work prompted him to express the opinion, according to Tanner, that bees appear to be excellent biological indicators of an atmospheric fluoride problem, since the presence of amounts greater than 1 microgram of fluoride per bee indicates permanent or intermittent fluoride levels which are higher than normal.

484. In summary, Professor Tanner stated that "it can be concluded that fluoride can be poisonous to bees. The toxicity varies with the age of the bee, the nutritional status of the bee, foraging activity, solubility of the [fluoride] compounds, the distance from the source of pollutant, nectar sources, wind direction and topography. Normally bees contain less than 1 microgram of fluoride in their bodies; 5 to 10 micrograms is normally accepted as the lethal dose, although some studies have shown higher concentra-

tions are occasionally found leading to speculation that a degree of resistance can be developed." (Vol. XI, p. 1614 to 1617).

D. FLUOROSIS IN CATTLE

(a) General Considerations

485. As Dr. Martin pointed out, it was the effect on animals in polluted areas which first attracted attention to the importance of industrial fluorosis. Many reports on this subject have been published during the past 50 years. The problem in the United States has been reviewed by Phillips and his associates (1960)[48], and in the United Kingdom a detailed investigation of the problem over an eight year period has recently been completed. Burns and Allcroft (1964)[51]; Allcroft, Burns and Herbert (1965)[52]. It is evident from the literature that cattle are the most susceptible of all livestock and, as in man, dental lesions are the first and perhaps the most sensitive sign of fluorosis. The age of the animal, as pointed out by Professor Willoughby, is extremely important in determining the presence or degree of fluorosis.

486. It is well known that, when animals are born from mothers which have been ingesting high levels of fluoride, the deciduous or temporary teeth of the newborn animal are perfectly normal. In order that dental lesions of fluorosis may develop, abnormally high levels of fluoride (e.g. 25 p.p.m. F) must be ingested after birth and during the period of enamel development, i.e. 9-12 months immediately preceding the eruption of permanent teeth. The first permanent incisor teeth of cattle erupt at about 18 months of age: thus it is apparent that an animal under 18 months of age will have normal teeth. For the same reason, if cattle are first exposed to excess fluoride when 4 years of age or over, their permanent teeth will have all erupted; consequently, no dental lesions of fluorosis will be observed, although other signs of fluorosis may be observed. Detailed descriptions of the eruption times, dental lesions of fluorosis in cattle, and abnormal wear patterns have been presented by Hobbs et al (1954)[59].

487. Reference has already been made to the study of *Fluorosis in Cattle* by Burns and Allcroft (1964)[51], as conducted on behalf of the U.K. Ministry of Agriculture, Fisheries and Food. Studying, as they were, the extent of fluorosis in livestock on 832 farms in 21 industrial areas of England and Wales, their findings and conclusions are of great significance. About dental lesions, they say:

> "These are the most sensitive index of absorption of abnormal amounts of fluorine, and levels of intake too low to produce

other symptoms can cause permanent changes in the enamel of the developing tooth. These changes are caused by replacement of the enamel lustre by a dull chalk-like appearance giving a mottled effect in the early stages, and by hypoplasia (defective or incomplete development) resulting in a thin enamel and small teeth which wear rapidly and irregularly, the enamel surface becoming roughened, grooved and eroded. Staining varies from slight yellow to dark brown and black; the colour depends on, and varies with, the diet and may not always accompany fluorosis. When staining occurs, it penetrates deeply into the defective enamel. Erosion or wear may vary from a slight clipping or blunting of incisal margin to severe wear down to the gums giving the appearance of the incisors having broken off or fallen out. Wear of molars can produce uneven levels with over-growth and prong formation which may interfere with normal grinding of the food. Excessive wear often causes exposure of the plup resulting in severe pain, especially when drinking. Dental effects are usually bilateral and occur only when the fluorine intake has been excessive during the period of development of the teeth. Temporary teeth are unaffected and the teeth of an adult animal remain normal if exposure occurs after the teeth are fully developed. Thus an adult animal with normal teeth may have severe skeletal and systemic fluorosis."

488. The *Fluorosis in Cattle* report continues:

"Since the extent of the changes depends largely on the length and degree of exposure to fluorine and since the permanent teeth calcify in a certain order, the pattern of dental abnormalities, especially when used in conjunction with other symptoms and clinical evidence, can be of great importance in the diagnosis of bovine fluorosis. Although dental abnormalities are characteristic, it should be emphasized that no single symptom is definitely diagnostic and Franklin [1950][60] has described dental lesions in sheep resembling those of fluorosis but caused by a calcium deficiency."

489. In the same connection, it is reported in the "Fort William Study"[17] that the incisor teeth showed mottling of the enamel, distortion and deformity. The cheek teeth showed excessive wear; by a process of selective abrasion the teeth had been so worn that long sharp points had formed which pressed into the gum of the opposing jaw. The interlocking of these prolongations prevented the animal from chewing its food effectively; particularly did this interfere with the process of chewing the cud. It was further reported

that "there was excessive wear of the cheek teeth, with ridging, grooving and the development of sharp prongs," and that "the incisor teeth were mottled and worn down to stumps." Comparable descriptions of the dental lesions, in cattle grazing on or consuming highly fluorine-contaminated herbage, are found throughout the literature and were supported by the evidence given at the hearings by Professor Willoughby and Professor Suttie.

490. Grazing sheep showed dental lesions almost identical with those shown by the cattle: on one farm in the "Fort William Study," 72 out of a group of 116 sheep (62 per cent), which were grazing in a field about 1,300 yards from the factory (fluoride emitting aluminum plant), showed severe dental abnormalities and a "similar state of debility" (to that of the cattle). On another farm, 39 out of 85 sheep (46 per cent), grazing about 1¼ miles from the factory, showed comparable lesions.

491. Of the many excellent and complete reports on the effects of ingestion of high fluoride-containing fodder on livestock, not one has recorded any "falling out" of teeth.

492. Several reports, dealing with dental lesions in sheep, have indicated that, as in cattle, pain was evident when some of the sheep drank cold water—obviously as a result of the cold water coming in contact with exposed sensitive parts of the worn teeth.

493. The literature, again supported by Professors Willoughby and Suttie, is consistent, recording a generally poor "condition" of dairy cattle which had been ingesting high fluoride content food, 40 p.p.m. or more, for long periods of time. Their coats lacked the normal lustre which might be expected, the skin and hair appeared to be on the "dry" side, and there was, in those animals with noticeable or severe dental lesions, loss of body weight. The young cattle, under two years of age, on the other hand, have been reported to be in quite good condition. The factor here is that, with marked wear and destruction of the teeth in some of the older animals, mastication was interfered with and an inadequate quantity of food was consumed. The affected cows deteriorated from malnutrition, as a secondary effect of the high fluoride content of the food; milk production, at this stage, naturally decreased. It is obviously important to recognize, as has been reported repeatedly in the literature, that the age of the cattle when exposed to the high fluoride-containing diet is a vital consideration. This may present some thoughts of significance in farm management in a so-called "polluted" area. It is therefore desirable to quote from a section of the "Fort William Study"[17] which deals with the detailed exam-

ination of an entire dairy herd of 45 cattle which grazed approximately one mile from the fluoride-emitting aluminum plant. The study is pertinent to our Port Maitland study and throws much light on the age and duration of exposure factors so relevant to compensation.

494. Production in this particular aluminum plant was started in 1929; there was a major expansion of the plant in 1938, and the plant reached maximum production in 1942. The examination of the herd in question took place in 1946. The report states:

> "No dental lesions were observed in the young stock (i.e. animals under two years old). A four-year old bull, which had grazed only for about a month, since September, 1943, had a perfect set of teeth. During the time the bull was housed on this farm, he was fed on roots, hay and corn grown on the farm, and received in addition such small quantities of artificial food stuffs as were available under the rationing scheme [wartime U.K. rationing for livestock]. When the adult cattle were considered in their age groups, certain significant points emerged. A 13-year old cow and an 11-year old cow had teeth that were normal for their age. Two 10-year old cows had teeth that showed slightly excessive wear. Of the five 9-year old cows, four showed definite dental lesions, whereas one did not; in one of the affected animals, it appeared from the involvement of the teeth in relation to their time of eruption that exposure to fluorine had begun about 7½ years previously. Three 8-year old cows were affected, though one did not appear to be badly affected; in one of the affected animals, the exposure to fluorine appeared to have begun about five years previously. All three 7-year old cows were affected and the exposure to fluorine appeared to have begun about five years previously. Both 6-year old cows were affected, but the permanent molars, that had erupted in the first year of life were not so badly affected as the teeth that had erupted later; this suggested that these two animals had not ingested herbage so heavily contaminated with fluorine in the first year of their life as in the second and subsequent years. Both five-year old cows were affected; in one, the exposure to fluorine appeared to have begun early in life; in the other, it appeared to have begun a little later. All five-year old cows showed marked dental changes. One three-year old milking heifer had a very badly affected mouth."

495. On an adjacent farm, in the "Fort William Study," two cows were examined. One, a six-year old purchased as a three-year old, had a normal mouth; the other, a three-year old reared

on the place, had dental lesions involving both incisor and cheek teeth.

496. Since little or no horse breeding is carried out in the Fort William area, mature work horses were generally purchased from outside the fluorine contaminated area. The observation that no fluorosis was observed in horses or ponies during the study may not be significant in spite of the fact that horses brought into the area obviously were fed for many years on locally-grown and hence contaminated hay, grain, etc.

497. As in the "Fort William Study," the M.R.C. (U.K.) study[17], the Wisconsin studies[48] likewise reported stiffness, lameness and swelling of the joints in affected cattle; other reports from Italy, the United States, Norway, Switzerland, etc., confirm these observations. The signs and symptoms of loss of "condition," debility and emaciation are comparable in most of the major reports. Exostosis and osteomalacia are to be found in severely affected animals and, as Willoughby and others have pointed out, spontaneous fractures, usually of the pedal bones, frequently result. Pain is usually evident in the affected leg; stalled animals are often seen with the foot resting on the edge of the manger. Several authors have made comments on the variability of bone lesions as found in cattle and sheep.

498. In *The Air Pollution Handbook* (1956)[46], the effects of various air pollutants on farm animals are described. In dealing with the symptoms of chronic fluorine poisoning, Phillips says:

> "Although in mild or marginal fluorine toxicosis, skeletal effects cannot be observed with precision, certain symptoms may be observed in advanced cases. A bony 'overgrowth' (exostosis) in advanced cases may be observed on the leg bones, jaw-bones, and ribs. Lameness may occur as a result of this overgrowth in the leg bones or of calcification of the ligaments in the legs. This may be followed by stiffness and calcification of the joints. Animals showing clinical fluorosis will invariably exhibit lack of appetite, emaciation and general ill health due to malnutrition, all of which will adversely affect milk production and retard growth. The systemic reaction of a fluorine-poisoned animal is primarily one of self-imposed starvation and the clinical symptoms seen, aside from the dental and skeletal effects, are identical with those of starvation. The systemic effects of excessive fluorine ingestion are not permanent, and they are readily amenable to dietary therapy with low fluorine ration. Mottled teeth remain unaffected and bony overgrowth little affected by such therapy."

499. Phillips' comments, of course, relate to the symptoms and pathological changes concomitant with chronic fluorosis as seen in sheep and cattle. Horses, hogs, poultry, etc., may develop comparable symptomatology at very high levels of fluoride intake; however, they have higher tolerance to fluorides and, since they depend much less upon roughage in their total food intake, are much less likely to develop chronic fluoride toxicosis.

500. Several symptoms of chronic fluorosis are duplicated in other forms of toxicosis, dietary deficiencies and even diseases. It is here that the chemical analysis for fluoride in the urine is a ready and useful aid in a differential diagnosis. Urine samples taken for fluoride testing should, for best results, be taken in the morning and, whenever possible, samples should be taken from several cows and pooled in order to give a reliable index of the urine fluoride content of the herd. Then, individual sampling becomes important and is fairly reliable, although variations do occur. For comparison, repeated sampling on individual animals is advisable. Many workers in the field have shown that a positive correlation exists, both in man and in domestic animals, between the amounts of fluoride ingested and the concentration of fluoride in the urine. Urinary fluorides are an admixture of fluorides ingested in food and water and fluoride released subsequently from the bones.

501. Publication 824—being a report of the Committee on Animal Nutrition of the Agricultural Board (U.S. National Academy of Sciences)[48]—states that:

> "Urinary fluorine concentrations are considered normal for the cow if they are below 10 p.p.m."

Suttie and his group found that the average concentration of fluoride in the urine of dairy cows increased within 2-3 days following the inclusion of NaF in the ration. Only in those cows where the urinary fluoride levels were 20-30 p.p.m. or more, for long periods, was there evidence of fluoride toxicosis. In this connection, the "M.R.C. (U.K.) Study"[17] indicated that the cattle in "contaminated" areas at Grantham and at Fort William showed fluorine in their urine in excess of 10 p.p.m. In Green's series of 4 heifers, the urine, corrected to a specific gravity of 1.030, showed fluoride levels of 26, 28, 69 and 28 p.p.m. respectively:

> "The contamination of the fodder at Grantham was shown to be literally enormous, a sample of grass containing 2,200 p.p.m. (dry weight) of fluorine."

502. The F ion is quite reactive and combines readily with calcium and phosphorus. It is thus understandable that most of the in-

gested F would find its way into the bones of the animal. According to Cass,[50] the F ions form hydroxyfluoroapatite by replacing principally hydroxyl (OH) and some bicarbonate (HCO_3) ions in the hydroxyapatite. The F as it occurs in bones is relatively insoluble and thus tends to remain there for long periods of time.

503. It has been proven beyond doubt that, quantitatively, the most definite measure of fluoride toxicosis is the degree of retention of fluoride in the body. Since about 95 per cent of the total fluoride retained in the body is to be found in the bony skeleton, according to Mitchell and Edman (1952)[49], the content of F in the bones is of the utmost importance in its relationship to the general health of the animal.

504. The concentration of skeletal fluoride depends upon the length of time of exposure, the level of ingestion, the type of compounds, and the age of the animal. Since most foods contain several p.p.m. of fluoride, the retention of fluoride in the bones continues throughout life. Not only do the bones of the older animals contain more fluoride than those of younger animals, but there is also a variation in the retention in various types of bones depending on their metabolic activity. Hence, as it has been pointed out, (Publication 824, National Academy of Sciences)[48]:

> "Cancellous bone, such as that in the skull, pubic bones, ribs and vertebrae, usually contain higher concentrations of fluorine than are found in the compact bones of the legs. The fluorine concentration in normal bones, generally lies between 100 and 1,200 p.p.m. of fat-free dry bone of mature cattle, or between 150 and 1,800 p.p.m. of ashed bone." . . . "the majority of fat-free dry mature cattle leg bones have between 400 and 800 p.p.m. of fluorine."

505. As long ago as 1939-40, Pierce[61] showed that the level of bone fluorine (on an ash basis) may increase 10 to 15 times above normal without any noticeable abnormal symptoms. He wrote:

> "The long latent period, a year or more in herbivora, during which the animal suffers no apparent untoward effects while steadily ingesting each day an amount of fluorine which eventually proves seriously toxic, may no doubt be explained by a gradual saturation of the skeleton until a stage is reached when its buffering effect is no longer capable of immobilizing the greater part of the absorbed fluorine, which is then free to exert general toxic effects."

Pierce has reported data indicating that fluorine in bones of sheep, ranging from 410 p.p.m. up to 5,900 p.p.m., was found in what

would appear to be normal bones, whereas a pathological diagnosis was usually associated with values of 7,000 to 8,000 p.p.m. So-called "normal" bones of hogs have been estimated to contain up to 3,000 to 4,000 p.p.m. of fluoride.

506. In the "M.R.C. (U.K.) Study,"[17] the normal range for cattle and sheep (fluorine as a proportion of bone ash) was considered to be from 100 to 1,500 p.p.m. They place values above 2,000 p.p.m. (in bone ash) as indicating absorption of abnormal amounts of fluorine, but "clinical symptoms, apart from mild to moderate tooth lesions, do not usually occur until values of over 4,000 p.p.m. are reached. In severely affected animals in Britain values of about 10,000 p.p.m. and upwards are often found and a few have been as high as 19,000 to 20,500 p.p.m., values which represent the approximate physiological saturation level."

(b) Diagnostic Considerations

507. There are several established diagnostic procedures described in the literature which are to be followed in establishing an irrefutable diagnosis of fluorosis in cattle. When these procedures are followed, one can, from the resulting data, also assess accurately the severity of the disease in an animal or herd so affected. These diagnostic procedures are:

(a) Chemical analysis of fluoride in:
   (1) diet or ration
   (2) urine
   (3) bone (or teeth)

(b) Tooth effects; i.e. chalkiness, mottling, erosion of enamel, enamel hypoplasia and excessive wear.

(c) Systemic evidence; i.e. lameness, anorexia, inanition, cachexia, exostoses and other bone changes.

508. Tooth effects or dental lesions in cattle with fluorosis can be described in quantitative terms following the widely used method of Dean, as outlined by Phillips et al (1960)[48]. This is as follows:

Rating	*Description*
0-1 Normal to questionable:	enamel smooth, translucent, tooth normal shape; slight changes of unknown origin, enamel flecks, chalky spots.
2 Very slight	slight mottling; very slight chalkiness* with or without stain.

3 Slight to moderate	enamel mottling, moderate chalkiness, erosion, teeth slight to severe staining, may be slight wear.
4 Moderate-marked	hypoplastic enamel, mottled, chalky, teeth show wear.
5 Excessive	enamel hypoplasia,** erosion, stained, and pitted in many cases; excessive wear of teeth.

*Chalkiness—Replacement of the enamel lustre by a dull chalk-like appearance. Mottling describes the pattern of chalkiness in the early stages. A horizontal pattern is suggestive of injury during formation of the tooth.

**Hypoplasia—Incomplete formation, i.e., a thin enamel; small tooth. Wear must be adjusted to the age of the tooth, and to the type of feeding.

509. Another system for grading the severity of fluorosis in cattle has been described by Burns and Allcroft (1966)[62]. Although the various grading methods use slightly different terms and different grade designations, they are generally in agreement. The two are listed here in the hope that they may be of value in further studies in Ontario.

510. Burns and Allcoft (1966)[62] use four grades as follows:

Grade I — Herds with dental lesions only, usually mild to moderate, and without apparent disability. Urine values up to 10-15 p.p.m. and bone values between 1,000 and 4,000 p.p.m. fluorine in bone ash. This is considered to be a "non-damaging" fluorosis.

Grade IA—"Damaging" dental fluorosis, which is of two types (a) when the abnormal appearance of the teeth resulted in a reduction of the selling price of the animals, even though there was not sufficient structural damage to the teeth to cause interference with feeding, (b) when dental damage was severe enough to interfere with feeding and result in loss of condition in animals but in which there was no lameness. This situation is well-known in fluorosis in sheep but does not seem to be common in cattle—lameness and stiffness being more frequently seen.

Grade II—Herds slightly affected with lameness, i.e., with only a few lame animals. Urine values 20-30 p.p.m. and bone values 4,000-6,000 p.p.m. fluorine.

Grade III—Herds severely affected with lameness, i.e., 10-30 per cent of the herd lame. Dental lesions and bony abnormalities well marked. Urine values 20-60 p.p.m. and bone values 6,000-10,000 p.p.m. fluorine.

Grade IV—Primary systemic fluorosis in which the fluorine intake was high enough to cause loss of bodily condition and a decline in milk yield during the first grazing season even in non-lame animals. Urine values often 60-80 p.p.m. or higher, bone values very high and up to 20,000 p.p.m. fluorine, with gross bony abnormalities in animals which were kept on the farm long enough for them to develop.

(c) Fluorosis in Ontario from Causes Other than Industrial Pollution

511. It is of singular interest to note that the condition of fluorosis, as found in some of the cattle in some of the herds in the townships of Sherbrooke and Moulton, is not unique to those townships.

512. It has been reported that a number of dairy herds in Ontario have been encountered which have shown symptoms of fluorosis where no industrial pollution was involved. In most affected herds, the sources of fluoride were mineral mixtures. In some instances the mineral mixtures were formulated with phosphorus supplements containing levels of fluoride such that the mineral mixes contained up to 10,000 p.p.m. of fluoride. (See also paragraph 464.)

513. One dairy herd which was affected with fluorosis had been fed a mineral supplement containing 10,000 p.p.m. of fluoride, while the drinking water contained 2 p.p.m. Even when the fluoride in the mineral supplement was reduced to 1,500 p.p.m., urine samples from the cows contained more than 10 p.p.m. and signs of fluorosis were observed. In other herds, fluorosis had occurred because mineral supplements containing fluoride levels of 2,000 p.p.m. had been fed in excessive amounts by the farmer.

514. The incidence of fluorosis in Ontario cattle appeared to be markedly reduced following the enactment of the federal regulations as to permissible maxima of F in feed mineral supplements. According to the present (1967) *Feeds Act* and *Regulations* a feed shall not contain fluorine in an amount likely to be deleterious to livestock, or exceeding the following proportions:

(i) 0.2 per cent (2,000 part per million) in any mineral feed represented for feeding direct to cattle or in any mineral feed for cattle containing up to 9 per cent of phosphorus;

(ii) 0.3 per cent (3,000 parts per million) in any mineral feed for cattle containing at least 9 per cent of phosphorus;

(iii) 0.6 per cent (6,000 parts per million) in mineral feed for swine; and

(iv) in a complete or balanced feed:

(a) 0.009 per cent (90 parts per million) for cattle and horses;

(b) 0.010 per cent (100 parts per million) for sheep;

(c) 0.014 per cent (140 parts per million) for swine and rabbits; and

(d) 0.035 per cent (350 parts per million) for poultry.

515. In spite of these regulations, excessive fluoride intake supplied through mineral supplements, feeds, water, or combinations thereof, is still a possibility; some farmers will continue to feed more mineral supplements than called for.

516. Industrial contamination with fluoride is not the only source of the element, and the hazard from industrial pollution can be exacerbated by feed, mineral supplement, and water. Good husbandry is a vital part of good farm management.

(d) Effects of Fluoride on Productive Performance of Cattle

517. Cass[50] has reviewed the effects of fluoride on different farm animals. Chronic fluorosis has an insidious onset, develops slowly, and involves primarily gross and microscopic changes in bone and teeth. Secondarily, there is progressive deterioration of the general health of the animal, dependent upon, or at least in association with, progressive changes in the skeleton and teeth. Under certain conditions, lameness develops and the animal ceases to graze because of the pain associated with movement or through loss of appetite, or perhaps for the two reasons combined. Individual animals may become incapacitated and die. The frequency of occurrence and the severity of the lesions vary widely between herds and individuals within a given herd.

518. Once established, moderate to severe dental damage induced by excessive fluoride may progress, because of continued stress upon the weakened and softened dental tissues. Such dental damage becomes important to the well-being of cattle, when affected teeth wear unevenly, or excessively, or break with undue ease, or become sensitive to cold water. Any of these conditions may lead to a seriously curtailed intake of food. But it must be repeatedly emphasized that, once a permanent tooth is fully formed and erupted, no significant change in its structure is induced by any level of fluoride intake.

519. One investigation of chronic fluorosis, with cattle moved into and out of pastures contaminated by fluoride-bearing wastes from industry, gives evidence of the rapidity and reversibility of the formation and re-solution of bone. Removal of the animals from contaminated feed resulted in an arrest of the fluoride-inducing resorption, while mineralization of the newly formed bone proceeded.

520. When the ingestion of fluoride by a seriously affected animal is terminated, the lameness and painful gait commonly diminish and disappear in a matter of days or weeks, only to reappear if the absorption of fluoride is resumed. When, however, extensive periarticular exostosis and ankylosis is present, lameness is generally permanent, even after withdrawal of fluoride from the diet. Dental damage in permanent teeth is irreversible.

521. The retention of fluoride tends to increase as the dietary level increases. Evidence shows that the retention may range from 12 per cent, at 30 to 40 p.p.m. in the diet, to 57 per cent, at 100 p.p.m. of diet. This may well be explained by the lower concentrations of dietary alleviators as the level of fluoride increases.

522. The apparent digestibility of nutrients, including dry matter, crude fibre, crude protein, nitrogen-free extract and other extract, has not been shown to alter in cattle and sheep ingesting feeds containing 108 and 208 p.p.m. F respectively, for long periods of time. There thus appears to be no direct effect of fluoride on utilization of feed by cattle and sheep.

523. The prolonged ingestion of elevated quantities of fluoride appears to have no direct effect upon conception, gestation and parturition, or lactation. However, lameness, dental damage, and loss of appetite, with an attendant loss in weight and markedly reduced lactation, may result, as a secondary effect, in the suppression or interruption of estrus, and perhaps in extreme cases a reduction of the weight of the newborn, and poor subsequent development of the young. The female animal would have to show manifest signs and symptoms of fluorosis before a secondary interference with reproduction could occur.

524. In cattle and sheep, the transfer of fluoride from affected mothers, through the placenta to fetuses or through milk to sucklings, is measurable but inconsequential and induces no recognizable changes in the young cattle and sheep. Analyses of bones and teeth from near-term fetuses, whose mothers had been subjected to ingestion of excessive quantities of fluoride, indicates that the

deposition of fluoride in the fetal skeleton is relatively slight and insufficient to cause dental or bony lesions.

525. Cass[50] summarized available information on the concentrations and dosages of fluorides consumed by farm animals, in association with their ages, the duration of exposure, and the occurrence of lesions. The data clearly indicate the variations in the response of animals to a given dosage, under the influence, no doubt, of a number of other factors. Because of these many variations, a condensation of the data to specific levels of dosage, in apparent casual relationships to signs and lesions, is likely to be highly misleading. For example, on the basis of F retention in the bones, urine excretion, weight gain and adult weight, and other factors, the tolerance of young dairy cattle to fluoride was less than 1.6 mg. F/kg. body weight daily; cows receiving 1.4 mg. F/kg. were in a marginal zone of toxicity. On the other hand, it was shown that 2-year old heifers on pasture could be fed up to 2.5 mg. F/kg. body weight for an extended period, with only mild pathological symptoms. The data showed that one of the most reliable measures of F toxicosis was the presence of skeletal F at levels in excess of 5,500 p.p.m.; interestingly, the cattle fed 2.5 mg. F/kg. of body weight showed a considerably lower bone concentration than this.

526. The evidence indicates that the average intake of 1 mg. of fluoride per kg. of body weight (as NaF or other equally soluble inorganic F compounds), ingested daily by cattle during the productive period of life is not associated with any demonstrable injurious or abnormal effect. This is about equivalent to having fluoride in the concentration of 30 p.p.m. in the diet of dairy cattle and 45 p.p.m. in that of beef cattle, with low levels in the water.

527. Suspected fluoride toxicosis may be diagnosed tentatively by the general health and condition of the herd and the presence or absence of dental fluorosis in the different age groups in the herd. However, other deficiencies can produce emaciation, lameness and discoloured teeth. Positive and supporting evidence of fluorosis, even in the presence of the characteristic lameness and dental damage, must be supplied by chemical data which indicate that concentrations of fluoride in the diet, urine, and bone, are above normal. Thus, clinical diagnosis supported by chemical analysis can positively identify fluoride toxicosis.

528. Once fluorosis has been diagnosed, the monetary loss through impaired production must be estimated for each animal in each herd on each farm, since in no two cases is the effect likely to be the same.

E. FLUOROSIS IN OTHER FARM LIVESTOCK

529. The same diagnostic procedures, where applicable, as followed in the clinical diagnosis of fluorosis in cattle, must be used in establishing an accurate diagnosis of fluorosis in other farm animals.

530. As mentioned previously, the order of susceptibility of farm animals to fluorosis is: calves, dairy cows, beef cattle, sheep, horses, swine and poultry (Allcroft et al, 1965)[52]. The feeding habits of these farm animals have received consideration in establishing permissible fluoride content of feed and mineral supplements (paragraph 514).

F. EVIDENCE FROM THE HEARINGS ON LIVESTOCK HEALTH

(a) Introduction and Evidence from Farmers

531. In previous paragraphs, a résumé of the effects of fluorides on livestock has been presented. Most of the area farmers, who were concerned with this problem on their own farms, were present at the hearings and heard the evidence of some, at least, of the consultants, and of their neighbours. This section deals specifically with evidence submitted at the hearings. The extent of "alleged" damage to livestock—and that adjective must be used at this stage in the report—had to be determined on evidence given by witnesses, from analytical reports of testing conducted by various persons in various government departments and agencies, from compiled statements presented prior to the calling of witnesses, from the records of the "Arbitrators" for the crop years 1965 and 1966, and from the records of "Claims" made for the crop year 1967 (which have not yet been assessed). Assistance was also gained from the submissions presented by Counsel for the "Air Pollution Committee" and by Counsel for ERCO.

532. Mr. G. Brooks, Counsel, represented some 32 farmers in the area under consideration. The evidence of the witnesses will be dealt with in the order in which they were called by their Counsel. Whenever it is feasible, and in order to stress particular points raised by witnesses or Counsel, the direct recording of evidence will be used.

(a) MR. TED McALONAN

533. Mr. Ted McAlonan farms about 70 acres, 2 miles NE of Port Maitland, within the northern part of the "cone" area. He stated that he maintains "a beef cattle and calf operation, and I

try to raise enough grain for my own use and possibly a couple of cash crops. Well, I have tried to keep about between 30 and 40 head of cattle, which consists of 10 brood cows, and raised the steers until they were two years old, and we also had yearlings which at times would be 40 head." Mr. McAlonan stated that the lameness of some of his cattle "always seemed to be more in the summer months than in the winter. In 1965, I had cattle for six weeks that just went to the field and laid down and, as far as I am concerned, never pastured. They just made it in and out." He also stated: "They did not want to eat this pasture at all with their teeth and, because of lameness, they weren't able to get around." He also stated: "Oh yes, I have lots of cattle that don't have any teeth, like their nipping teeth. Well they had nippers, about 8 teeth in the front and those teeth—by '65 some of these cattle didn't have any teeth at all, they were gone, but the roots appeared to be there, but they were rubbed right off as if they had turned soft, and I don't think these cattle could pasture."

Mr. Brooks: *(p. 707)* *Now, I take it from what you said that you had some difficulty with the health of your animals. Is that so?*

Mr. McAlonan: *That is right.*

Mr. Brooks: *When did you first notice that?*

Mr. McAlonan: *In 1963.*

Mr. Brooks: *And can you describe it to the Commission?*

Mr. McAlonan: *Well, all had lameness and cattle crippled up. They didn't seem to want to pasture when they went to the field. And in 1963 and 1964, we didn't know what was wrong, but in 1965 it was diagnosed as fluorosis and . . . I had, I would say, about half of my brood* [cows] *that was crippled up, finding it hard to get to the field and back.*

Mr. Brooks: *What did you do with those cows?*

Mr. McAlonan: *Well, I took them* [in] *in February of 1966, after the opportunity was there in 1965, and inside on silage they did recover some and I thought that I should get rid of them because they had the best salvage value that they had in two or three years, so at that particular time I sold them.*

Mr. Brooks: *Did you replace any of your herd from 1963 onwards?*

Mr. McAlonan: *No.*

Mr. Brooks: *And, when you finally disposed of those cattle, can you assist the Commission by describing the quality of them or the prices of them?*

Mr. McAlonan: *Well, they were back in a lot better fashion* [condition] *than they were in the summer of 1965; they seemed to come around on silage and good feed through the winter, like lots of greens. And I got these cattle back to, well, I would say, an average type of cow as far as weight was concerned, and I had had such trouble for two years or three years I thought this was the time to dispose of them, when they were a little bit of salvage value.*

Mr. Brooks: *Now, when you received the compensation, you were, I take it, permitted to keep any salvage on the cattle, is that so?*

Mr. McAlonan: *That is right.*

Mr. Brooks: *Were you satisfied with the compensation paid to you?*

Mr. McAlonan: *Oh, I am satisfied, it was average.*

Mr. Brooks: *Did you think you got less or more than you were entitled to or you felt you deserved?*

Mr. McAlonan: *Well, I felt the compensation in keeping the salvage was somewhere near the figure it should have been, but it really upset your operation. When you get rid of good cows that are ready to have a calf drop, you sit every year with no chance to replace it, and this wasn't taken into consideration.*

Mr. Brooks: *Did you actually have any cattle die on you?*

Mr. McAlonan: *I never did have any die.*

Mr. Brooks: *Did you have a veterinarian diagnose any injury or illness in your cattle?*

Mr. McAlonan:	*No, when they came to check my cattle, I guess it would be in the fall of 1965, or possibly early 1966, this was the only time that I had brought him for the purpose. . . .*
Mr. Brooks:	*What are your feeding methods for your cattle? Do you grow some of your own feed?*
Mr. McAlonan:	*I grow all my own feed.*

534. Such a description of lameness, damage to teeth, and difficulty in pasturing, together with evidence of recovery while feeding on silage in the barn, is certainly most suggestive of fluorosis. If the fluoride concentration of the urine of the affected cattle had been determined and had been in excess of 20 p.p.m., the diagnosis would have been positively established. But there is no evidence to show that urine samples of affected cattle were taken and therefore no results are available. Nor are there records or other evidence to indicate how many of the "30 or 40 head of cattle" were affected or to what degree. Nevertheless, on the basis of Mr. McAlonan's testimony, it is accepted that some of his cattle certainly had fluorosis and, on the basis of the compensation claims agreement, awards were made for the whole herd—even though perhaps only a portion of the cattle was affected. No evidence as to the ages of the cattle in the herd is available, even from compensation records.

535. The difficulty in obtaining specific and factual evidence—and this is simply to illustrate a point and is in no way being mistrustful of the individual farmer—may be illustrated by this evidence:

Mr. Brooks: *(p. 709)*	*Did you replace any of your herd* [30-40 head] *from 1963 onwards?*
Mr. McAlonan:	*No.*
Mr. Brooks: *(p. 708)*	*And when you finally disposed of these cattle. . .*
Mr. McAlonan: *(p. 709)*	*. . . I thought this was the time to dispose of them.*
Mr. Brooks: *(p. 713)*	*What about your cattle in 1967?*
Mr. McAlonan:	*Well, I have raised heifers since 1965 and those cattle at present are arriving at 3 years old. . .*

Mr. Lamek: (p. 715) *Mr. McAlonan, I think you said that you had 30 to 40 cattle prior to 1965?*

Mr. McAlonan: *That is right.*

Mr. Lamek: *And now you have twenty-six?*

Mr. McAlonan: *I have 26 at present, yes.*

Mr. Lamek: *Does this include the calves and yearlings?*

Mr. McAlonan: *Well, as yet my '68 crop, none of these cattle have come in yet.*

Mr. Lamek: *But you expect to have cows* [calves] *from some of them?*

Mr. McAlonan: *Yes.*

Mr. Lamek: *And the total of 26 will increase?*

Mr. McAlonan: *It will probably go to 31 or 32 or something like this.*

Mr. Lamek: (p. 716) *But you didn't replace the cows that you had sold?*

Mr. McAlonan: *I sold about half my herd in February of 1966, after I had received my compensation. I have replaced these cattle but I did not do it at that particular time.*

Mr. Lamek: *Does that mean that you did not replace them all, that is, all the cows you disposed of?*

Mr. McAlonan: *Well, I have raised my heifer calves from 1965; those cattle are brood cows now.*

536. Mr. McAlonan was asked by Mr. Brooks, Counsel for the "Air Pollution Committee," about the effect on cattle breeding.

Mr. McAlonan: (p. 714) *Well, this is definitely causing me some concern; we seem to have trouble getting cows in calf. I have never had any trouble with cattle not reproducing, but they don't reproduce in one year like they should. I have lost practically a season for this. In fact, my young heifers now, none of them are bred.*

Mr. Brooks: *Have you consulted a veterinarian or anyone with regard to that?*

Mr. McAlonan: *I haven't, no.*

Mr. Lamek: (p. 721) *Just one final matter, Mr. McAlonan. This business of your cows breeding—did I understand you to say that, in February of 1966, you sold four cows that were in calf?*

Mr. McAlonan: *Yes.*

Mr. Lamek: *Did you have any trouble breeding those?*

Mr. McAlonan: *Well, it is like I said before, these cattle have a tendency to take a long time to get a calf, but I have never had any problem with cows not being able to be bred or get in calf. But the time is the most important thing here ...*

Mr. Lamek: (p. 222) *How do you breed your cows, is it artificial?*

Mr. McAlonan: *I have kept a bull of my own up until last year and since I have had this trouble I got rid of my bull, and this affected cattle and this type of thing.*

Mr. Lamek: *Did you have more success this year with breeding, since you got rid of your bull?*

Mr. McAlonan: *I have really had no problem artificially. Those cattle are all in calf at present.*

(b) MR. RIENTS BOORSMA

537. Mr. Rients Boorsma operates with a brother, father and mother, two farms, one about three miles and the other two miles E of Port Maitland, within the southern part of the "cone" area. They are full-time dairy farmers, with a herd of approximately 72 head.

Mr. Brooks: (p. 736) *Now from 1960 onward, would you explain to the Commission what, if any, health problems you have had with your herd?*

Mr. Boorsma: *In 1960, I did not know of any problems at that time, but in 1962 I think it was our neighbour Mr. Casina who started to figure that he was having problems, and at that time we were not too concerned yet.*

Mr. Brooks: (p. 737) *Now, when did you first notice difficulties and what were those difficulties?*

Mr. Boorsma: *In 1965 we noticed our first difficulties to some extent that we could see.*

Mr. Brooks: *And could you tell us what you noticed?*

Mr. Boorsma: *Yes, the cows were getting slow. It was a big problem at first, they were getting you might say lazy. They would not pasture properly and if we did give them some new pasture they didn't want it and then a severe drop in milk, which was worse in 1965, but it started in 1961.*

Mr. Brooks: (p. 738) *Are you able to tell the Commission in percentage approximately how much it dropped having regard to the number of your herd?*

Mr. Boorsma: *Yes, I would say in 1964 it dropped about 15 per cent maybe.*

Mr. Brooks: (p. 739) *They* [the veterinarians] *diagnosed it as fluoride poisoning.*

Mr. Boorsma: *Yes.*

Mr. Brooks: *Were any of your cattle sent away or were they just tested.*

Mr. Boorsma: *At that time they were just tested.*

Mr. Brooks: *What happened to your herd in 1965?*

Mr. Boorsma: *We had a severe loss of milk at that time and when we sent the cows out, and I have got to figure this out, because we kept them inside one year. In 1965 they were outside and they would be walking crippled, and this may be out of 50 that we were sending out at that time, maybe about 10 would show signs of lameness.*

Mr. Brooks: *Is this just in walking or in any other way?*

Mr. Boorsma: *No, when they were standing in the stall and being milked, they were lifting their front feet up considerably, lifting one up and then the other, just like it was bothering them, and when they were walking out they would be dragging their feet and stopping and you would have to chase them out. When you get them out in the yard you would have to chase them to get them out in the field. When they got out*

	in the field, why they would eat for a little while maybe and then they would lay down. They were always laying down if they could get away with it. In fact, right after milking, they would lay down again, and this is what showed up their sore feet, I would say.
Mr. Brooks:	*Did you notice anything about their teeth during this period of 1965?*
Mr. Boorsma:	*Just what the vet told us, that they were infected with fluorides.*
Mr. Brooks:	*Now, I just want to know what you noticed, just tell us what your own observations of your herd were, Mr. Boorsma?*
Mr. Boorsma:	*Well, I must say that in 1965 I was not too awfully interested in those teeth.*
Mr. Brooks:	*Was there anything, or did you notice anything about joints or anything* [of] *that nature?*
Mr. Boorsma:	*Yes, the joints would get swollen up quite severely in some cases, and the bottom part of their feet would get swollen up, and I would say that their toes would get awfully hard, instead of breaking off naturally they would stay on and we had to have people come and take them off, which cost money.*
Mr. Brooks:	*What about their appetite?*
Mr. Boorsma:	*In 1965, their appetite was very poor.*
Mr. Brooks:	*Now, turning to 1966, what further developments or changes were there in that year, if any?*
Mr. Boorsma:	*I would say conditions in 1966 were much the same as in 1965. We had got rid of some cows because the factory paid for some, the factory paid for practically all of the cows.*
Mr. Brooks:	*That is the Electric Reduction Company in 1966 paid for the whole herd, did they?*
Mr. Boorsma:	*Yes, that is right.*
Mr. Brooks:	*On the basis of a total loss?*

Mr. Boorsma: *Yes.*

Mr. Brooks: *And did you, in fact, dispose of the herd?*

Mr. Boorsma: *I would say maybe about 20 per cent.*

Mr. Brooks: *Did you still have any of these cows in 1966 or do you still have some from 1966 that you were paid for by the company?*

Mr. Boorsma: *I think we still have some, yes.*

Mr. Brooks: (p. 745) *. . . between 1964 and 1965, you estimated your production loss at about 15 per cent, is that so?*

Mr. Boorsma: *That is correct.*

Mr. Brooks: *From 1965 to 1966 it was a further 15 per cent?*

Mr. Boorsma: *No, we raised it again, it went up when we kept them inside and we had better production of at least 15 per cent and maybe 20 per cent.*

Mr. Brooks: *And between 1966 and 1967, what happened?*

Mr. Boorsma: *We had a loss of about 20 per cent again.*

Mr. Brooks: *How is your production going now?*

Mr. Boorsma: *Compared to a healthy herd, I would say that we run about 15 per cent below normal.*

538. In January 1968, ERCO notified the farmers of the area whose hay had been "tested" for fluoride content that, in those cases where the concentration in the dry hay exceeded approximately 25-30 p.p.m., the Company would destroy the remaining quantity of hay and replace it with hay purchased from outside the area. Mr. Boorsma testified in this manner:

Mr. Brooks: (p. 746) *Has your hay been replaced?*

Mr. Boorsma: *Our hay was replaced, the little bit that we had left. They notified us that the fluoride count was too high and ERCO came around and said they would burn it and replace it.*

Mr. Brooks: *When did you receive this notification?*

Mr. Boorsma: *I don't know the date, but I would say late in January of 1968.*

Mr. Brooks:	*And your herd has been working on this hay supply since when?*
Mr. Boorsma:	*Since last August.*
Mr. Brooks:	*And it was practically all gone by that time?*
Mr. Boorsma:	*Yes, I think we had around 15 tons or 20 tons left.*

539. Mr. Boorsma stated that "in 1965 we had quite a problem with calving and especially again with heifers now, but in between that it had been about the same." Since 1965, he had become more conscious of the effects of fluoride on the teeth and, in 1966 and 1967, "checked the cows frequently and I found there were many cows that had brown teeth. They are supposed to be all white, and several missing, but other than that . . ." Mr. Boorsma was asked, "Have you actually had any cattle die on you?" Mr. Boorsma said, "We had one die out in the field from kidney infection." (p. 749), and, "We had another cow, well, I wouldn't say it didn't actually die, but it was the next thing to it, it went crazy." Although Mr. Boorsma mentioned that they had three cattle die, no information was given about the third animal. (It might perhaps be well to note here that the mortality in dairy herds in Ontario is approximately 1 per cent or a little lower per annum. Thus Mr. Boorsma, with a 72 head dairy herd, might have expected to have some cows die in a 3-year period.)

540. With inadequate or no records of cattle purchased or sold, of milk production, except for one year, etc., it was virtually impossible to determine the actual number of cattle from the 1965 herd, compensated for in 1966, which were sold, which were retained in the herd, which had produced calves, and how many were actually still in the herd in January, 1968. And, although Mr. Boorsma stated that about 10 of the 50 cows showed lameness in 1965, he was compensated for the whole herd. The following passage is relevant:

Mr. Lamek: (p. 759)	*Do I understand then that the whole herd, an award was made for the whole herd in 1965?*
Mr. Boorsma:	*Yes.*
Mr. Lamek:	*For 50 cows, 4 yearlings and 9 calves?*
Mr. Boorsma:	*Yes.*
Mr. Lamek:	*Did you replace any of these 60-odd cows?*

Mr. Boorsma:	*Yes.*
Mr. Lamek:	*Approximately how many?*
Mr. Boorsma:	*About 9 or 10.*
Mr. Lamek: (p. 763)	*What about the rest of those cows?*
Mr. Boorsma:	*I would say about 40 cows left over.*
Mr. Lamek:	*Do you still have them?*
Mr. Boorsma:	*I would say some of them, yes.*
Mr. Lamek:	*How many of them?*
Mr. Boorsma:	*I would say around 20 maybe.*
Mr. Lamek:	*What happened to the other 20?*
Mr. Boorsma:	*They were sold later.*
Mr. Lamek:	*The 20 you still have, are they producing milk?*
Mr. Boorsma:	*It is just that we can't afford to sell them all and get new ones; like I said, they cost too much money to buy.*
Mr. Lamek:	*That is not what I asked, I asked if the cows that you had from that 40 are still producing milk?*
Mr. Boorsma:	*Yes.*
Mr. Lamek:	*Have you had calves from them?*
Mr. Boorsma:	*Yes, some calves.*
Mr. Lamek: (p. 764)	*What is the size of your herd now?*
Mr. Boorsma:	*I think close to 72 or 73.*
Mr. Lamek:	*That means you must have bought some cows in the meantime.*
Mr. Boorsma:	*No, we raised some cows, and we buy some, yes.*

(c) MR. JACOB VANDERBEEK

541. Mr. Jacob Vanderbeek maintains 108 acres, 2½ miles NE of Port Maitland, within the north-central part of the "cone" area.

He has since disposed of a herd of around 28 milking cows (Holstein). His son-in-law, Mr. A. Bruinsma, acted as his interpreter.

Mr. Brooks: *(p. 807)*	*Approximately what is the present size of the herd?*
Mr. Bruinsma:	*The herd is completely gone.*
Mr. Brooks:	*When did he get out of the dairy farm or when did he get rid of his herd? How was it disposed of?*
Mr. Bruinsma:	*He got rid of the dairy in January, 1967.*
Mr. Brooks:	*How many head of cattle did he have then?*
Mr. Bruinsma:	*He had at that time 23 cattle.*

542. In evidence it was stated that Mr. Vanderbeek's "problem started in 1963. In 1965, he was quite down already." At that time, he had "about 27 milking cows. Two or three cows were sold for salvage prices." In describing the condition of these cows it was said, "The cow was lame, it had swollen legs and would not eat properly; he was awfully skinny and practically no meat on him at all any more." The teeth were described as "black." One of the disabled cows was taken to "... Guelph and from there on it came out that the illness was fluoride." This was on July 9th, 1965. Other cows in the herd were being affected to the extent that, "In 1965, we got three cows shot in the field. They could not get up anymore." Loss of milk production was stated as severe—a drop from a "10 can milk contract on a daily basis to a 6½ can to 2½ can" or from about 800 pounds per day to less than 200 pounds per day. In 1966, compensation for 1965 was paid for the "loss of animals," "22 cows and one dead, $225.00 for that dead one." In other words, the herd in 1965 had been reduced to 23 head. Mr. Vanderbeek received compensation at $225.00 per head for the whole herd.

543. It is necessary at this time, so that the next piece of evidence becomes meaningful, to state that Mr. Vanderbeek was not in good health, had been examined on several occasions by his physicians and had been in Toronto General Hospital under the care of Professor Wightman, who had diagnosed Mr. Vanderbeek as having bronchitis and emphysema, osteoarthritis of the spine and phlebitis in one leg. There was no evidence of fluorosis.

Mr. Brooks: *(p. 830)*	*May I put it another way, would he still be in the dairy business today if his herd had not been injured?*
Mr. Bruinsma:	*No, he would not.*

Mr. Brooks:	*Would he have gotten out at the same time?*
Mr. Bruinsma:	*He would have sold his farm.*
Mr. Brooks:	*What I am getting at is, did he stop the farming operation because of the herd damage or because he couldn't carry on any longer?*
Mr. Bruinsma:	*His health, he couldn't carry on any longer.*

(d) MR. LEONARD McINTEE

544. Mr. Leonard McIntee operates 330 acres, 1½ miles NE of Port Maitland, within the north-central part of the "cone" area. He owns 35 acres and rents the remainder. As he said, "At the present time, I am a corn and hog man" having been raising hogs since 1947. In 1960, he had 24 sows and "had a complete operation from farrowing to market." Mr. McIntee stated, "Well, it was in 1965 really that we had really bad reproduction, and caused me a lot of trouble." He stated that "from 7 sows I had 7 pigs." Prior to 1964, he claimed that he was producing on the average of 15 or 16 pigs per sow per year. He figured that if this problem had not been present, he would have had 400 hogs for sale. "Well, you can figure 24 sows would give you at least 20 pigs [per sow] and so make claim for compensation for that number." Compensation was paid for lost or absent litters—for "208 piglets that were not even born."

Mr. Brooks: *(p. 840)*	*In 1965 you experienced a reduction?*
Mr. McIntee:	*I experienced a terrible reproduction problem.*
Mr. Brooks:	*Was it smaller pigs or smaller weights or what was it?*
Mr. McIntee:	*Premature births and very small litters, and puny pigs.*
Mr. Brooks:	*Was there anything else?*
Mr. McIntee:	*I had lost three sows, and I had to sell 6 other ones.*
Mr. Brooks:	*Did you have any difficulty in breeding?*
Mr. McIntee:	*No trouble in breeding, but just the reproduction that caused me problems.*
Mr. Brooks:	*In 1965, did the same conditions continue?*
Mr. McIntee:	*Yes.*

Mr. Brooks: *What did you do about it?*

Mr. McIntee: *I just stayed with my smaller number of the sows, 15 at the present time, hoping ...*

Mr. Brooks: *There were 15 in 1965?*

Mr. McIntee: *Yes.*

Mr. Brooks: *Were you getting the same average production?*

Mr. McIntee: *No, I was having smaller litters.*

Mr. Brooks: *Were some of the litters born dead, or what?*

Mr. McIntee: *Well, they were so near dead you might as well have called them dead.*

Mr. Brooks: *Have you ever been advised that fluoride had any effect on your hog operations, and if so, by whom and when?*

Mr. McIntee: *I never have been told that.*

Mr. Brooks: *Have any of your animals been sent away for examination?*

Mr. McIntee: *No.*

Mr. Brooks: *You received compensation for these hogs, did you?*

Mr. McIntee: *I did.*

Mr. Brooks: *And in what years was that?*

Mr. McIntee: *For 1966.*

Mr. Brooks: *And there was nothing for 1965?*

Mr. McIntee: *No.*

Mr. Brooks: *Are they up for compensation in 1967, do you know?*

Mr. McIntee: *No.*

Mr. Brooks: *Is there any reason?*

Mr. McIntee: *Well, the conditions have been a little better.*

Mr. Brooks: *You have noticed an improvement, have you, in 1967?*

Mr. McIntee: *Yes.*

Mr. Brooks: *Have the litters been increasing?*

Mr. McIntee: *Yes.*

Mr. Brooks: *Are they back to normal?*

Mr. McIntee: *Yes.*

Mr. Brooks: *Do you grow your own feed?*

Mr. McIntee: *Yes, I do.*

(e) MR. KEITH WRIGHT

545. Mr. Keith Wright owns 80 acres and rents an additional 50 acres. He lives within the north-central part of the "cone" area, 2½ miles NE of Port Maitland. He works full-time at the Sherbrooke Metallurgical Company plant in Port Maitland. Mr. Wright raises hogs and had, at one time, 120 of them on his farm. In 1965, he had "either 25 or 27" hogs. He stated that in "the latter part of '65 and '66, sows didn't produce. Maybe one to three pigs, either born dead or maybe two or three not far from being dead." It was, as he said, primarily a problem with his sows. "Not so much in the fattening hogs. The sows would get overly fat and you could feed them on fresh air and cold water, they would still get fat and they won't have any pigs." When asked by Mr. Brooks what steps he took to overcome these difficulties, he answered, "I called the vet and he said there is nothing that I can see wrong except maybe your sows are a little too fat and maybe you haven't been feeding them the right thing. But I have been feeding the sows as usual but maybe they were overfed, I don't know."

546. Mr. Wright also stated (Vol. VI, p. 860), "My older sows, the ones that were pastured outside and being fed concentrates on the outside, they produce nothing but dead pigs. The younger sows, the ones I kept inside, of which they were seven, and that I raised myself, I bought feed too, they averaged me 11 a-piece and there is the difference." Subsequently, Mr. Wright called the veterinarian in to see "a couple of growing hogs" which had "died recently" and "he came up with an entirely new disease altogether, one that I had not heard of, and he called it Mulberry Heart Disease. I suppose pigs are the same as people and they get these things too." His chief complaint about the compensation which he received was that the numbers in a litter were based on a county average. He felt that his operation is above average and that his sows would average 11 pigs per litter "on five litters every two years."

(f) MR. EARL DEAMUDE

547. Mr. Earl Deamude has an oil distributorship and is interested in farming "as a kind of hobby." He farms 85 acres, 1¾ miles NE of Port Maitland, within the north-central part of the "cone" area. He has a mixed farm, some crops and some animals. Buying his first registered bred heifer in the fall of 1962, he purchased two more in 1963—all three being safely in calf—and built up his herd to eight in 1963. One calf was born dead; and "one [cow] didn't carry her calf through." He had no calves from any of his cows in 1964. The oldest cow that he had "went lame and refused to eat pasture. She would stand and beller and she would act almost wild." Mr. Deamude bought his hay and concentrates, but pastured his animals on his own land. When one of his old animals "had shown signs of lameness, she had long hoofs" and he trimmed these and "it still didn't clear up her lameness, swollen fetlock, the same as was shown in the slides yesterday afternoon [by Professor Willoughby]. I decided there was possibly something wrong with her so I should get rid of her and, when I put them in the barn in the wintertime I fed them hay and concentrate, and her lameness—the lameness seemed to disappear to a degree—but in the spring I put them back out on pasture and again she wasn't out there three weeks before she started to get lame."

548. Speaking of the general condition of his animals, which were being fed "concentrate kind of grain quite heavily," Mr. Deamude pointed out that, "You couldn't get a sleek look on these animals like they normally do when they are being fed the way these animals were being fed. Their hair was dull and you couldn't get a good body finish on it." Mr. Deamude indicated that, "I only had one animal with lameness," and in mid-July, 1966, "I disposed of all the animals I had. It was not economically possible to keep them there under the conditions that were existing."

(g) MR. RUSSELL ELKOW

549. Mr. Russell Elkow farms with three brothers in a partnership known as Willowdale Farms. The Elkow Brothers farm some 456 acres, 2½ miles NE of Port Maitland, within the north-central part of the "cone" area. They have been in the dairy cattle business since 1947 or 1948, having kept a herd of about 35 milking cows during the period 1960-1965. In August, 1966, they disposed of their dairy herd and presently are in cash crops and beef cattle, which herd they started in September, 1967. From the dairy herd, they still have one milking cow, six young heifers, and twin calves.

550. The dairy herd was in good health until 1964, when it was noticed that the cattle went "downhill" when they were put out to pasture in the spring, "and in the fall they were a pretty rough looking bunch; their flesh wasn't there."

551. The age of the dairy cattle varied "from three years up to maybe ten years or twelve years." The condition of the cattle—"in the wintertime when we brought them back in, we were pushing the chop to them and they really increased." The milk production of the herd "did not go down bad until June 7th, 1965."

Mr. Gordon: *What happened then?*
(p. 950)

Mr. Elkow: *On June 7th, we got this here blast from ERCO.*

Mr. Gordon: *Could you explain that?*

Mr. Elkow: *Well, it was that bad that we couldn't see our neighbours on both sides.*

Mr. Gordon: *It was something that was in the air then?*

Mr. Elkow: *Yes.*

Mr. Gordon: *How long did that last?*

Mr. Elkow: *It lasted for an hour or an hour and a half.*

Mr. Gordon: *What did it look like?*

Mr. Elkow: *Just like a heavy fog.*

Mr. Gordon: *Was it white in colour?*

Mr. Elkow: *Yes, white.*

Mr. Gordon: *What happened?*

Mr. Elkow: *In three or four days later, our milk production fell bad.*

Mr. Gordon: *Do you know how much it fell?*

Mr. Elkow: *I think it was around 300 or 400 pounds.*

Mr. Gordon: *How long did the milk production stay down?*

Mr. Elkow: *It gradually kept going down and we started feeding heavily and it started to come back again.*

Mr. Gordon: *When was that?*

Mr. Elkow: *Well, we started right after the milk was going down, we started feeding them heavily on chop.*

Mr. Gordon:	*Was it a matter of days or weeks until production was back to normal?*
Mr. Elkow:	*Just a matter of days, but it never did get back to normal.*
Mr. Gordon:	*How close did it come to being normal?*
Mr. Elkow:	*It was around 200 or 300 pounds.*

552. Mr. Elkow indicated that "those blasts" occurred both in winter and in summer and roughly about once each week. He identified "it" as coming from a stack at the ERCO plant and not from the lagoon area. He indicated, too, that the cattle pastured well in 1964 and up to June, 1965. Since obtaining the beef cattle in September, 1967, Mr. Elkow had used the same pasture as he had used for this dairy herd.

Mr. Gordon:	*And have you noticed any difference so far as pasture is concerned?*
Mr. Elkow:	*Well, we were pasturing over six heifers, oh, right from spring. And we got the beef cattle in there. They like it.*
Mr. Gordon:	*They were eating the pasture?*
Mr. Elkow:	*Oh, yes.*
Mr. Gordon:	*And do you think conditions improved then during the 1967 season?*
Mr. Elkow:	*I think so.*
Mr. Gordon:	*Do you have any complaints at the present time?*
Mr. Elkow:	*No.*

Going back to Mr. Elkow's dairy herd (Vol. VII, p. 955-956.):

Mr. Gordon:	*Now, what was the reason that you disposed of your dairy herd?*
Mr. Elkow:	*Well, in '65 when we got that blast on June 7th, our cows were going downhill. In the winter there, well, we had trouble. They couldn't get up. And then when we let them out to pasture in May of '66 then they really went downhill. It took about a month and they really went downhill. They were crippled right up.*

Mr. Gordon: *When you say that they went downhill, you mean that they became lame or crippled or what?*

Mr. Elkow: *Yes, they became lame, yes.*

Mr. Gordon: *Did they have any other symptoms?*

Mr. Elkow: *No, the lameness.*

Mr. Gordon: *Were they able to walk?*

Mr. Elkow: *Most of them were able to walk, yes.*

Mr. Gordon: *Did any of them actually lie down?*

Mr. Elkow: *Yes, we had two or three which we sold to this drover, and we had to drag them on the truck with the tractor.*

Mr. Gordon: *Did any of your cattle die?*

Mr. Elkow: *No.*

Mr. Gordon: *Did any of your cattle have trouble giving birth to calves?*

Mr. Elkow: *No.*

Mr. Gordon: *Did they breed normally?*

Mr. Elkow: *Yes.*

Mr. Gordon: *No reproductive problems?*

Mr. Elkow: *No.*

Mr. Gordon: *So that insofar as your dairy herd was concerned, the only thing you noticed was that they seemed to go downhill health-wise?*

Mr. Elkow: *Yes.*

Mr. Gordon: *And then they became lame?*

Mr. Elkow: *Yes.*

Mr. Gordon: *How many of them were lame?*

Mr. Elkow: *Well, you could say about half of them. The ones that did get lame, we used to keep them in the barn and, say, in a couple of weeks, they came out of it and then we would let them back out in the field.*

Mr. Gordon: *Did you have any problems with any of your calves?*

Mr. Elkow: *No.*

Mr. Gordon: *None at all. And the compensation that you received in 1965 and in 1966, that would have been for your whole dairy herd at that time, is that correct?*

Mr. Elkow: *Yes.*

Mr. Gordon: *And were you satisfied with the compensation that you received?*

Mr. Elkow: *Yes.*

Mr. Gordon: *Did you feel that the amounts awarded were fair?*

Mr. Elkow: *Yes.*

553. The question of cattle allegedly dying as a result of fluorosis was posed to each of the witnesses. Mr. Elkow's answers to questioning on this subject is cited, as has been done in the other instances:

Mr. Gordon: *(p. 959)* *Have you ever seen any cattle dead in the fields surrounding your farm?*

Mr. Elkow: *No.*

Mr. Gordon: *Have you ever heard of any of your neighbours or farmers in the area having dead cattle on their farms?*

Mr. Elkow: *Yes, I heard about it.*

Mr. Gordon: *How many? How many dead cattle have you heard about?*

Mr. Elkow: *I wouldn't know, I just heard about this. It wasn't my concern.*

Mr. Gordon: *Do you know whether it was one or five or ten?*

Mr. Elkow: *One or two, that is about all.*

Mr. Gordon: *Do you know of any farms in the area where there were more than one or two cattle that were reported to have died?*

Mr. Elkow: *No.*

In cross-examination by Mr. Brooks, Mr. Elkow stated that none of his dairy herd had, to his knowledge, any teeth trouble.

Mr. Brooks: *(p. 966)*	*Did any of your cattle, to your knowledge, have any teeth trouble, the dairy cattle?*
Mr. Elkow:	*No.*
Mr. Brooks:	*Pardon?*
Mr. Elkow:	*I don't think our cattle had any teeth out.*
Mr. Brooks:	*Any of your dairy herd cattle that had any teeth problems?*
Mr. Elkow:	*No teeth problems.*

554. It was recorded that Mr. Elkow and his brothers kept one cow "and one bull," as well as some heifers, from their original dairy herd and have pastured them with their newly acquired beef cattle.

Mr. Lamek: *(p. 968-9)*	*I am interested in this one cow—from your original dairy herd, is it?*
Mr. Elkow:	*Yes.*
Mr. Lamek:	*Why did you select that particular one, sir, to keep when you got rid of the rest?*
Mr. Elkow:	*Well, she just happened to be fresh at that time.*
Mr. Lamek:	*But she had been lame?*
Mr. Elkow:	*Yes.*
Mr. Lamek:	*And she was pastured all this year?*
Mr. Elkow:	*Yes, in 1967.*
Mr. Lamek:	*And you say she is now in A-1 condition?*
Mr. Elkow:	*Yes.*
Mr. Lamek:	*She is not limping now?*
Mr. Elkow:	*No.*
Mr. Lamek:	*How is her milk production?*
Mr. Elkow:	*Good.*

(h) MR. ERNEST PUHL

555. Mr. Ernest Puhl farms 100 acres, 3½ miles SE of Port Maitland, just at the boundary of the "cone" area. He carries on

a mixed farming operation with grade Holstein and Herefords. The Holsteins were kept for the raising of veal calves. He did not sell milk on a commercial basis. In 1960, Mr. Puhl had a herd of some 25 head, increasing it to 32 head in 1966. His barn was struck by lightning on June 14th, 1966, and "during the summer there I had to sell some of my cattle because I didn't know whether I could rebuild or what I was going to do."

Mr. Gordon: (p. 1032) *Did you grow your own hay?*

Mr. Puhl: *Yes, I did.*

Mr. Gordon: *Is that what you are feeding them?*

Mr. Puhl: *I was feeding my own hay and my own silage at the time.*

Mr. Gordon: *Were you feeding them any grain besides?*

Mr. Puhl: *Yes.*

Mr. Gordon: *It was the grain that you had grown?*

Mr. Puhl: *Yes it was.*

Mr. Gordon: *Are you a full-time farmer?*

Mr. Puhl: *Not now I am not, no.*

Mr. Gordon: *When did you stop being a full-time farmer?*

Mr. Puhl: *Well, after I had all this here damage and stuff,* [I] *had to go and get a job so I could make payments on my barn.*

Mr. Gordon: *And up to that time, so far as your cattle were concerned, you observed that some of them were lame, is that correct?*

Mr. Puhl: *Well, during the summer of '66, in the fall, towards the fall.*

Mr. Gordon: *Prior to that time you hadn't noticed anything abnormal about this?*

Mr. Puhl: *No, I did not.*

Mr. Gordon: *And what did you do with the lame cattle?*

Mr. Puhl: *Well, during the summer there when some of the cows were coming in well, the drover came along; he bought ten off me.*

556. In connection with compensation, further examination followed:

Mr. Gordon: (p. 1035)	*And was the compensation that was awarded to you fair in your opinion?*
Mr. Puhl:	*Well, for those which I was compensated for, I figure it wasn't too bad, but at that time I had these steers on my property which should have been sold a year ago and I did not receive any compensation for these steers.*
Mr. Gordon:	*Now, out of the cattle that you were compensated for, were you allowed to retain them on your property?*
Mr. Puhl:	*Could you ask that question again, please?*
Mr. Gordon:	*Well, you received some compensation for selling your cattle, is that correct?*
Mr. Puhl:	*That is correct.*
Mr. Gordon:	*Were you allowed to keep these cattle or were they taken away from you or subject to compensation?*
Mr. Puhl:	*No, I got a slip right here saying that I was supposed to retain the money which I received from the cattle.*
Mr. Gordon:	*You could keep the cattle for which you had been compensated, right?*
Mr. Puhl:	*Yes.*
Mr. Gordon:	*And then did you in turn sell those cattle?*
Mr. Puhl:	*Yes, I did.*
Mr. Gordon:	*And you obtained additional money for them?*
Mr. Puhl:	*Yes. I have one cow there and one steer left out for a purpose.*

557. Mr. Puhl, having rebuilt his barn, "bought eleven steers this fall [1967] and I have two cows which I replaced, and one cow that I kept, and one steer. I have fifteen head altogether." Since he "built this barn for the purpose of tying-in 41 head of cattle, and I have 11 box stalls besides that," he hopes that the pollution will be cleared.

558. In cross-examination by Mr. Lamek, Mr. Puhl said that he had sold some of his cattle in 1966, after his barn had been struck by lightning and he had nowhere to house them.

Mr. Lamek: (p. 1939)	*But your reason for selling them was that the barn had been struck by lightning?*
Mr. Puhl:	*That is right.*
Mr. Lamek:	*You say you saw nothing abnormal in your cows between 1960 and 1966?*
Mr. Puhl:	*No, I didn't.*
Mr. Lamek:	*How old would your cattle be in 1966?*
Mr. Puhl:	*Some were seven and eight years old.*
Mr. Lamek:	*How long had they been on your farm?*
Mr. Puhl:	*I raised them.*
Mr. Lamek:	*You raised them from calves to whatever age they happened to be in 1966?*
Mr. Puhl:	*That is right.*
Mr. Lamek:	*And, in that time, you didn't observe anything abnormal?*
Mr. Puhl:	*No.*

Mr. Puhl also claimed that he had difficulty in raising chickens, and a few "years back I used to always get about 150 chickens and they would get so big [about a year old, he said later] and they just die off, and I never could find out the reason why." Mr. Puhl had not sought any professional advice relative to his chickens and stated that no examinations had been carried out on them. He does not keep chickens now because "they would only die off anyway, so I figured what is the use in keeping them." Later, Mr. Puhl stated that he permitted them to "run outside," presumably to forage, and later testified when asked, "How many of them actually died?" that, "I would say about 50 per cent of them." He sold the remaining 50 per cent, he said.

(i) MR. FRED JONKE

559. Mr. Fred Jonke farms 70 acres, 3½ miles NE of Port Maitland, in the north-central part of the "cone" area. He has, at the present time, 18 cows and 12 heifers, mixed grade Holsteins, Guernseys and Ayrshires. In 1960, he had "about 20 head" of cattle.

He noticed nothing wrong with his cattle during the period from 1960 to 1965 but, in 1966, "I noticed a few of my cattle were lame." This occurred in the spring of the year "as soon as I turned them out" to pasture. Mr. Jonke disposed of 13 cattle, "I started right in '66 . . . as soon as they got bad I sent them away," and purchased three replacements. In 1966, an award, for the year 1965, was made for 22 grade cows, 7 two-year old heifers and five younger heifers. He is still dairy farming, with 18 cows milking.

Mr. Brooks: (p. 1053) *Did any of your cattle die from this condition that you have described?*

Mr. Jonke: *No.*

Mr. Brooks: *And you heard the evidence that the other farmers had given about the condition of their cattle?*

Mr. Jonke: *Yes.*

Mr. Brooks: *So far as your cattle are concerned, is it just the fact that they went lame, is that all that you know about them?*

Mr. Jonke: *Definitely.*

Mr. Brooks: (p. 1054) *There is no other condition that you could describe about them?*

Mr. Jonke: *No.*

Mr. Brooks: *They didn't have any swelling joints or anything wrong with them?*

Mr. Jonke: *No, I didn't see any.*

(j) MR. JOSEPH CASINA, SR.

560. Some of the problems allegedly experienced by Mr. Casina have been presented in the previous chapter, under "Crops and Vegetation." Simply that it may be recorded in this chapter, it is noted that, in a written statement, as read by Mr. Casina at the hearings (Vol. IX, p. 1353, lines 4-8), he says, "Had a cow drop a calf prematurely, also had a cow give birth to a cripple; cattle were overlooked as tests were not made until 1965, then they were down with fluorosis." From the reports of claims and awards, it is noted that Mr. Casina was awarded damage for one cow in 1966 and that he had submitted a claim in 1967 for damage for one cow.

(k) MR. ROBERT PAISLEY

561. Mr. Robert Paisley farms 230 acres, 2½ miles N of Port Maitland, within the northern part of the "cone" area.

562. Until the spring of 1967, he carried on dairy farming. Since then, he has grown grain crops—mostly corn.

Mr. Gordon: (p. 1180) *What were the reasons that you got out of dairy farming?*

Mr. Paisley: *Well, I had my cows tested last spring and they said they had fluorosis.*

Mr. Gordon: *I see, and so you got rid of the herd; is that correct? What sort of farming do you now carry on on this farm?*

Mr. Paisley: *Well, I have to, I guess, go to corn mostly.*

The remainder of Mr. Paisley's evidence had reference to crops and has been dealt with in that chapter of the report.

(i) MR. JOHN CARRUTHERS

563. Mr. John Carruthers farms 130 acres, 2½ miles NE of Port Maitland. He works in Dunnville five days per week. The only evidence given about his cattle indicated that he got "out of the cattle business in 1966" because "of the problems with the cattle" (Vol. VIII, p. 1208, lines 11-18).

564. Mr. Carruthers was asked about his pigs:

Mr. Gordon: (p. 1212) *What was your experience with your pigs?*

Mr. Carruthers: *Well, we have a sow who will have a litter of pigs and you will get about five different sizes. I blame that from feeding the corn to the pigs.*

Mr. Gordon: *Did you have these pigs examined by anyone to determine whether the sow, as such, had fluorosis or any disease resembling fluorosis?*

Mr. Carruthers: *There were two veterinarians there from Guelph.*

Mr. Gordon: *Yes, and what did they do?*

Mr. Carruthers: *They just looked at them and made out a report and chased them around the pen and what they wrote down I have no idea.*

Mr. Gordon: *Did you ever hear from them again?*

Mr. Carruthers:	*No.*
Mr. Gordon:	*Did you ever see or get a copy of that report?*
Mr. Carruthers:	*No.*

(m) MR. HOWARD RITTENHOUSE

565. Mr. Howard Rittenhouse has been a farmer on the same farm of 110 acres, 3½ miles E of Port Maitland, for over 50 years. The property lies within the southern part of the "cone" area. He has been in the dairy business for over 30 years, with, at the present time, some "42 or 43" Holsteins. His evidence relative to livestock is reproduced here:

Mr. Gordon: *(p. 1224)*	*Now, during the last, say, 7 or 8 years, could you tell us what your experience has been insofar as farming is concerned and what possible problem, if any, you have had?*
Mr. Rittenhouse:	*Well, I don't know what you mean by that question.*
Mr. Gordon:	*Well, this hearing is concerned about allegations concerning pollution. Do you feel that your farm has been affected in any way and, if so, would you tell us?*
Mr. Rittenhouse:	*Well, I don't know. I haven't seen anything around our place. We feed the crops we grow and, fortunately, we have never had any trouble with our cattle going lame or loss of production or anything of that kind, and I think according to what I hear around here we have been very fortunate. I have no explanation for it; I don't know why or anything about it.*
Mr. Gordon:	*Have you had any reproductive problems with your cows?*
Mr. Rittenhouse:	*Not any more than I ever had in my life.*
Mr. Gordon:	*Did you ever raise any pigs?*
Mr. Rittenhouse:	*Well, I used to have some pigs.*
Mr. Gordon:	*Have you had any in the last 7 or 8 years?*
Mr. Rittenhouse:	*Oh, yes, but we haven't had any for around a couple of years now.*

Mr. Gordon: *Well, you haven't had any around for the last two years; that would be 1966 you got out of pigs?*

Mr. Rittenhouse: *Yes, but not on account of this pollution business.*

Mr. Gordon: *Now, in the period from 1960 to 1966, did your sows have any problems?*

Mr. Rittenhouse: *Not any more than usual.*

Mr. Gordon: *Nothing more than usual?*

Mr. Rittenhouse: *No. I say again that I think we must have been lucky.*

Mr. Gordon: *Do you know to what you could attribute that fortune?*

Mr. Rittenhouse: *Which?*

Mr. Gordon: *What would be the reason for that?*

Mr. Rittenhouse: *I have got no idea. I haven't any more idea than you.*

Mr. Gordon: *All right. That is quite true. It is just that in this area, I mean, you have neighbours to the south of you and to the west and to the north.*

Mr. Rittenhouse: *Yes, true, true.*

Mr. Gordon: *They all seem to have problems?*

Mr. Rittenhouse: *Yes, true, that is why I tell you that I think we have been very fortunate.*

Mr. Gordon: *All right.*

Mr. Rittenhouse: *I have no explanation whatsoever.*

566. In order to bring out evidence relative to compensation for his cattle, it is necessary to introduce here evidence about Mr. Rittenhouse's crops:

Mr. Gordon: *(p. 1228)* *Could you tell us anything whatever about the crops that you grow and what your experience has been with them, or is that the same?*

Mr. Rittenhouse: *Well, we have had no difference in any crops. They may be loaded with this here trash, whatever it is, but as far as the growth is concerned, I have never seen anything at all.*

Mr. Gordon: *I see.*

Mr. Rittenhouse: *We filled our silos off the acreage year after year; we have a good crop of hay, around a half to three-quarters of a ton per acre, and I am satisfied with the crops.*

Mr. Gordon: *And is your farm under-drained?*

Mr. Rittenhouse: *Not tiles—we have a few tiles, but not many.*

Mr. Gordon: *And you have always been a full-time farmer on this farm?*

Mr. Rittenhouse: *Yes, always.*

Mr. Gordon: *You haven't worked part-time in the city or in any industry?*

Mr. Rittenhouse: *No.*

Mr. Gordon: *Have you ever had to make a claim for compensation for damages?*

Mr. Rittenhouse: *Well, I did but I didn't have to.*

Mr. Gordon: *Could you explain that, please?*

Mr. Rittenhouse: *Well, I think I can. In the first place, our cattle has never been tested. I think it would be two —I guess it is two years ago this spring that Dr. Nelson was there from Guelph and tested either two or three, I am not sure which, but we never heard tell of it.*

Mr. Gordon: *You mean you never got the results of the tests?*

Mr. Rittenhouse: *No, we never got a report of it. We have had tests taken of hay and grain and fodder, they come and get it, but we never heard no tell of it, so along in, oh, somewhere—I don't remember just when it was— I can't remember the time—just a minute: It was along in the summertime and a fellow came in there one day and he turned out to be an arbitrator, so we talked a little while and there was nothing said about it, and along later in the fall—we never put a claim in—along later in the fall he came in there one day and, talking this*

thing over, and, well, we told him, "We've got no claim in and there is no report from this test that Dr. Nelson took of these couple or three cows." "Oh," he said "I have got that," and I forgot what he told me. There was none of them high. One was nine, I think it was, one for 13, and the other one was a little over. He says, "What do you want to do?" and I says, "Nothing," but he wanted us to put in a claim for our whole herd of cattle and scrap the whole works. "Well," I says, "we have got a business." "Well," he says, "you could go into beef cattle." "Well," I said, "I don't want to do that." So we did some business back and forth and finally he said, "Well, how be it if we claim ten?" "Well," I said, "we never have had no trouble with our cows, we have never had cows go lame or anything of this kind." "Well," I said, "maybe they have it, but I don't know anything about it." But I said we never had any, but he said "You sell cows occasionally?" and I said, "Sure, we have sold four or five cows already this year, but not for that reason." We sell a cow if she happened not to get a calf and there are some different things. I don't [meddle] *around; I sell them. Now, maybe that accounts for the reason we have never had a lot of lame cows. But I don't know much about that thing; but I can understand when a cow is lame.*

Mr. Gordon: *You would cull your herd from time to time, if an animal didn't look as though it would stay in shape?*

Mr. Rittenhouse: *That is right, yes. We had a meeting up at the hall and I found out that these cattle had to be out of the stable in 30 days.*

(n) MR. EDWIN HARVEY

567. Mr. Edwin Harvey and a brother operate a dairy farm of "about 55 cows and heifers" on 175 working acres, 2½ miles E of Port Maitland. They are full-time farmers. His evidence was confined to crops.

(o) MR. L. NIECE

568. The Niece brothers operate a 140-acre farm, 1 mile SE of Port Maitland. They grow cereal grains and carry on some beef cattle operations. It was only in incidental evidence that Mr. Niece indicated that "our cattle didn't seem to want to eat pasture . . . we would take them and change them in another field, just the same, and it would be the same thing." They still have beef cattle on the farm but, "Well, they don't look as good as they used to."

(p) MR. PETER PURYCH

569. Mr. Peter Purych farms a small plot 1½ miles SE of Port Maitland, "Just a small farm and I just grow—like wheat, hay and cattle." Part of his evidence is reproduced here:

Mr. Gordon: (*p. 1287*) *What was it you wanted to tell us?*

Mr. Purych: *Well, it is not only that, you see, first, before I heard people speak about pollution, and I had never been aware of that, I was kind of, like, at a loss on it in the beginning.*

Mr. Gordon: *When did you first hear about this pollution?*

Mr. Purych: *Well, it was in '63, I believe, and I don't believe it myself at that time, and again it just make me interested that I go to a meeting one time that a farmer had called on that purpose, so I hear that and I still just go for the interest; not for the complaint, but then representative from the Council sent some doctors from Guelph and they test my cattle in the barn, and I find that I get letter from the Department of Health afterwards that all my cattle is affected and I have to dispose of them.*

570. Subsequent to evidence presented on alleged crop damage and alleged water pollution, reference was made again to his cattle:

Mr. Gordon: (*p. 1293*) *Generally speaking, how do you find farming in this area? Do you have a fairly successful farming operation?*

Mr. Purych: *Yes, because I think I have always been a farmer, always, but lately, as I say, I have to dispose of my cattle and I just lost my feeling to go ahead and start over.*

(b) Evidence of Dr. Nelson on Area Animal Health Problems

571. It would be anticipated that, if lameness developed in a substantial number of cattle in an area, the local veterinarians, perhaps unacquainted with the ailment, would, through the Departments of Agriculture and Health, call for the services of a clinical veterinary consultant from the Department and from the University of Guelph. In September, 1966, Dr. F. C. Nelson of the Veterinary Services Laboratory, Guelph, visited the area. Subsequently, in 1967, Dr. Nelson again visited the area. The evidence of the hearings shed considerable light on the problem (Volume X, beginning at page 1423).

Mr. Gordon: *What were you expected to do and what, in fact, did your department do?*

Dr. Nelson: *We did exactly what we were asked to do—we were asked to simply indicate, through the examination of animals and the collection of urine, those premises which we considered had a higher than normal level of fluorine.*

Mr. Gordon: *And how many premises did you visit?*

Dr. Nelson: *I think it was 20—19.*

Mr. Gordon: *. . . what were the general conclusions insofar as the cattle were concerned?*

Dr. Nelson: (p. 1424) *We found evidence of fluorine on some farms and, on others, no evidence.*

Mr. Gordon: *Were any of the cattle from the area shipped to either your department or to Guelph for examination?*

Dr. Nelson: *There were two dead cattle arrived at the Department of Pathology at the Ontario Veterinary College, University of Guelph, in the winter* [1967-1968].

Mr. Gordon: *They were dead on arrival?*

Dr. Nelson: *Dead on arrival, yes?*

Mr. Gordon: *And what was done with these cattle?*

Dr. Nelson: *They were subjected to necropsy procedure and were found to have died from grain overload.*

Mr. Gordon: *Grain overload? What gave them grain overload?*

Dr. Nelson: *Consumption of too much grain at one time.*

Mr. Gordon: *Was there any evidence of fluoride poisoning?*

Dr. Nelson: *No, there was no evidence of fluoride poisoning.*
(p. 1425-26)

Mr. Gordon: *I think your report here concludes that there was evidence of fluoride poisoning in some cattle?*

Dr. Nelson: *Yes.*

Mr. Gordon: *Could we deal briefly with swine, and tell us what your experience has been with that particular animal in these three townships?*

Dr. Nelson: *Well, trying to determine whether a premises supporting swine was suffering from a problem is very difficult. We couldn't collect urine. I was concerned about that. The principal complaint seemed to be one of reproduction and I wrote to Dr. Crampton, who is Professor Emeritus of Nutrition, MacDonald College, and inquired about fluorosis and its effect on reproduction and he reported that, to the best of his knowledge, fluorine poisoning had no effect on reproduction. However, this seemed to be the report we received from every owner of swine in the area and we included this, the premises with swine on, in the premises we considered affected, but this was entirely on the owner's opinion.*

Mr. Gordon: *So you had no personal knowledge or were not able to conduct any tests to obtain evidence that it did have an effect on swine?*

Dr. Nelson: *No, we were not.*

Mr. Gordon: *Insofar as sheep are concerned, what was your experience in these three townships?*

Dr. Nelson: *We examined sheep on one premises, of Niece brothers, and we found evidence both of dental lesions and bone from fluorosis.*

Mr. Gordon: *When you say you examined the bones, these would be on dead animals?*

Dr. Nelson: *No, these were live animals. It is a clinical examination. We saw lameness and we saw deformities.*

Mr. Gordon: *Did you conclude that these sheep were affected in some way by fluoride?*

Dr. Nelson: *Yes, we did.*

Mr. Gordon: *Insofar as hens and poultry are concerned, what did you find?*

Dr. Nelson: *We examined two flocks of poultry. These were not poultry that were being kept in normal housing, as we consider poultry housing to be, but were chickens that were running outside, eating worms and bugs and living off the land, so to speak, and they had bone lesions, in that some of the poultry that we saw had enlargements of the bones of the leg, exostosis; and in order to determine if they were affected, we sacrificed two chickens on each premises and dissected out bones and submitted them to the Air Pollution Laboratory, but they are not able to run a test on the bones.*

Mr. Gordon: *So far as poultry is concerned, do they suffer from any other disease which might affect the bones?*

Dr. Nelson: *Yes, there is a common disease of poultry called Leukosis, and one form of this disease affects bone.*

Mr. Gordon: *Is that fairly prevalent in poultry today?*

Dr. Nelson: *(p. 1431)* *Quite prevalent, yes.*

Mr. Brooks: *And, out of the 19 premises that you visited, in how many did you find evidence of fluoride intoxication in the animals?*

Dr. Nelson: *First of all, you have to establish a level. We established a level. We established that, on the basis of literature available, a level of 15 p.p.m. could be considered positive and 10 p.p.m. suspicious.*

Mr. Brooks: *This is urine?*

Dr. Nelson: *Urine, yes, and so we considered anything that we got animals on* [premises] *with a level of over 10 p.p.m. as affected premises. This would be on 14 premises.*

Mr. Brooks: *Fourteen premises. Is it a fair way of putting it that there was evidence of clinical significance of fluoride intoxication?*

Dr. Nelson: *Yes.*

Mr. Brooks: *Are you prepared to say that, on the remaining premises, there was no evidence of fluoride intoxication in the animals, or is it that there just may or may not have been and you were unable to find it?*

Dr. Nelson: *No, I think that the latter would be true, that there may or may not have been. Mind you, this involved a two-day visit and therefore I would imagine that there could have been animals that had evidence of fluoride intoxication that we did not pick up. This is certainly not an intensive examination, nor the examination of every animal on every premises, either.*

Mr. Brooks: *I do notice that you say, Dr. Nelson, in your brief, "In my opinion, sows on the McIntee farm were affected." Again, are you basing your opinion on what Mr. McIntee saw?*

Dr. Nelson: *This, and the location of the premises in relation to premises with cattle. There was a dairy herd in the direct line from the plant. Robert Paisley had affected animals on his premises. He was almost in a direct line with the plant and a little further away, and Mr. McIntee reported that he was feeding entirely home-grown produce, so this was simply a matter of an assumption on the basis of circumstantial evidence.*

Mr. Lamek: *(p. 1436)* *I think you said that you were asked to indicate, through an examination of the animal and through analysis of urine, those premises which had animals on them which seemed to have had a higher than average ingestion of fluoride.*

Dr. Nelson: *Yes.*

Mr. Lamek: *Does that mean, Dr. Nelson, in your mind, those animals which had been damaged by fluoride?*

Dr. Nelson: *No, we were not asked to establish damage. It was very clearly pointed out that, because an arbitrator was involved, we were to establish this on the basis of premises, rather than numbers of animals or degree of involvement or damage.*

Mr. Lamek: *In that case, wouldn't you agree that perhaps 10 p.p.m. is rather low as an indication of any kind of damage, 10 p.p.m. in urine?*

Dr. Nelson: *I don't think that you could take a urine reading and associate this with damage at all, that is number one. Number two, I think that there is a considerable variation in urine. This is why you don't go on a farm and take one animal. There is a considerable day-to-day and time-of-day variation in your findings and, as a matter of fact, Gardiner, whom I quote, "Gardiner's Veterinary Book on Toxicology," recommends that a number of samples be taken so that you don't declare a farm an "affected" farm on the basis of one sample.*

Mr. Lamek: *Right, and I take it that is what you took, one sample from each animal?*

Dr. Nelson: *Of a representative number, you might call it a random screening.*

Mr. Lamek: *You didn't go back and take samples at a later stage?*

Dr. Nelson: *We did not.*

572. The decision to conduct the survey—one cannot call it a study—in the manner in which it was undertaken, was arrived at during a meeting held on May 8th, 1967, attended by Dr. Worton, Mr. Drowley, Mr. McLoughry and Dr. Nelson. Dr. Nelson in his "Report on Activities of the Veterinary Services Branch in relation to the Problem of Fluorine Poisoning in Livestock on some farms in the Dunnville area" (1966-67), stated:

> "At this meeting I mentioned that collection of urine should be used as a herd screening test rather than a diagnostic procedure for each animal . . . consequently, I advised that urine be collected from the minimum number of animals on any one premises that would provide significant findings and that these animals, if possible, be those that had developed demonstrable tooth and bone lesions or lameness."

573. The determination of "affected premises" was based, obviously, on a cursory examination of a few cattle in 19 herds, and on individual urine samples from those selected cattle. The study of a pollution problem, wherein damage to livestock and crops was a major consideration, deserved more than a two-day "random screening" effort.

574. Sympathetic as one might be in going through this type of evidence, one cannot excuse the obvious lack of knowledge about the problem of fluorosis as it affects livestock, the criteria necessary for its adequate diagnosis, the age factors involved, the relative susceptibility of species, the diagnosis on a "premises" basis, etc., etc. All of which has been thoroughly reported in the scientific literature.

575. And, of course, Dr. Nelson's statement that "Swine being the most susceptible of animals, acute poisonings frequently involve this species" (from his report), is quite incorrect. However, returning to the evidence recorded at the hearings, further light is shed on the inadequacies of the survey:

Chairman: *(p. 1442)* *Doctor, I would like to ask one or two questions. On these herds which you inspected, what percentage of the cattle of all ages within any given herd would you classify in the four grades of fluorosis?*

Dr. Nelson: *At the time that we examined these cattle—you mean with respect to dental lesions?*

Chairman: *The four grades, and grade two* [one] *has two divisions.*

Dr. Nelson: *At the time we examined these animals, we didn't know that there were four divisions for dental lesions; we merely looked at these animals not as . . . and I read Dr. Suttie's evidence, not in the way he examined them, but more in terms of clinical examination. We saw lesions, from a very slight mottling to actual hypoplasia of teeth or excessive wear of teeth, of both incisor and molar teeth, but we did not classify them.*

Chairman: *What percentage of any one "affected herd" might have shown lameness or real damage to the teeth?*

Dr. Nelson: *Well, lameness seemed to affect a very small number of animals, and the effect seemed to vary.*

> *For instance, Fred Jonke had a cow down nearly all summer, to a degree that she had a lot of pressure sores on her body, and I saw this cow in September* [1967] *and she was on her feet and seemed to be eating normally; and yet an animal that had absolutely no evidence of ever having been lame—the farmer had not noticed any signs of fluorosis at all—would have very bad dental lesions. The number of affected animals in this way varied in herds, but I would think—I saw rather fewer signs, clinical signs involving teeth and lameness and bone deformity and this sort of thing, than I expected to see.*

576. It should be noted that Mr. Brooks, Counsel for the local "Air Pollution Committee," had several persons give evidence on the damages and losses, insofar as the alleged pollution affected livestock, and other witnesses concentrated on the crop and vegetation aspect of the problem. Thus, the meagre evidence on the "Health of Animals," as given by Mr. Casina, Mr. Paisley, Mr. Carruthers and others, does not imply that they did not suffer damages in this respect. Likewise, since the evidence of Mr. Deamude, Mr. Vanderbeek, Mr. Boorsma and others, was basically confined to the "Health of Animals" aspect of the problem, it should not be inferred that they did not necessarily experience crop or other damage.

577. The evidence of Mr. Purych (paragraph 569), who as a member of the audience requested permission to give evidence, reflects the attitude of despair and frustration engendered in some of the area residents where—from their own experience they had noticed nothing wrong with their cattle and then on the basis of a sample or so of urine taken from an unknown number of cows, by an unknown person, and without knowledge of the results and apparently without discussion—they are advised that "their cattle are contaminated" and the herd must be "disposed of." Such action is sheer callousness and completely unscientific.

578. At least two farmers claimed that they grew all their own feed—Mr. McAlonan with a beef cow-calf operation and Mr. McIntee with a pig enterprise. Perhaps these men actually do purchase a mineral or a mineral vitamin pre-mix to help balance out their home-grown feed, but they do not consider this is a part of their "feed." Certainly, without such precautions, Mr. McAlonan might be expected to encounter lameness and emaciation from deficiencies of such nutrients as phosphorus and vitamin A. It is to be hoped that Mr. McIntee would also be purchasing a protein sup-

plement to mix with his home-grown grain, otherwise the overfat condition he described in his sows, together with their failure to reproduce normally, could readily be attributable to a deficiency of protein and many other critical nutrients present in commercial supplements, which are made specifically for the purpose of balancing out home-grown grain supplies.

579. It has been stressed in this report that the diagnosis of fluorosis in cattle cannot justifiably and adequately be made on the basis of a sample of urine from this cow and that cow. Repeated single or 24-hour samples of urine, taken from individual animals or a pooled sample from several cows, may, if a higher than normal fluoride concentration is found, suggest that fluorosis is present. If "typical" lameness is present in an animal or in several of the animals, then that evidence, too, may suggest that fluorosis is present. If "typical" dental staining and tooth damage is found in the mouth of an animal, that, too, suggests that fluorosis may be present. If it is known that the animal has been consuming "contaminated" food, over a period of many months and even years, this, too, may suggest that fluorosis may be present. And if a bone sample is analysed and a higher than normal fluoride content is found, that indicates that the animals have, over a long period of time, ingested high concentrations of fluoride. But no one clinical sign or symptom, and no one chemical test, can indicate that an animal had, at that time, fluorosis. More than one clinical sign or symptom must be associated with an acceptable chemical test. If the F content of urine is the criterion, the fluoride concentration must be demonstrated to be higher than normal on more than one occasion.

580. To claim that "cattle are contaminated," or that a herd be "disposed of," on the basis of the evidence presented at the hearing, without much regard to the age of the animals, or how long they had been in the area, and on the manner of the examinations conducted, is, in our opinion, unwarranted. That there has been fluorosis, in some of the cattle on several of the farms in the area, is unquestioned. There certainly has been some loss of cattle from the ingestion of above-normal amounts of fluoride over a long period of time, but how many? There definitely has been "lameness" of some cattle—there has been severe lameness in some cattle; there has been evidence of dental fluorosis in some cattle; there has been, in some cases, substantiation from fluoride analyses of urine and fodder, and there has been the word of veterinarians, government officials, and good and honest farmers in the area, that fluorosis has been present. But this does not indicate that

fluorosis was present in the herds, either to the extent or to the degree that one would be led to believe from some of the evidence and from some of the claims and awards for compensation. That this, in our opinion, is the case, is not to place onus on the doorstep of any one group—the farmer, the arbitrators, government officials, or the industries, or even on the terms of reference of the awards. If blame there has to be, we would say that it was a combination of inadequate organization to handle such a problem, lack of knowledge on the part of many persons (even when such knowledge was available), an appalling lack of communication, an early unawareness of the potential seriousness of the problem, delays and procrastinations, lack of effective co-ordination at the right stage, and confusion involving multiple legislative and jurisdictional authority.

G. EVIDENCE ON EFFECT OF FLUORIDE ON REPRODUCTION IN ANIMALS

581. In the study, "Fluorosis in Cattle," Allcroft and associates (1965)[52] stated that, with reference to reproduction:

> "The only significant abnormality was an incidental mycotic abortion. Fertility, as assessed by length of calving interval, number of inseminations per pregnancy and pregnancy rate to first insemination in animals born at Fenton Manor, was within the normal range."

Udal and Keller (1952) did associate infertility with fluorosis in cattle, but the evidence obtained from reports by Hobbs and his colleagues (1954)[59], Mitchell and Edman (1952)[49] and others, indicates, without much doubt, that fertility is not affected until the level of fluoride intake is sufficiently high, and extended over a sufficient length of time, to interfere with food consumption and the general condition of the animal.

582. During the hearings, Professor Suttie, a consultant for ERCO and an eminent research worker in the field of fluoride nutritional studies, was questioned (Vol. VII):

Mr. Lamek: *(p. 980)*	*Now, you were in this courtroom yesterday and you heard certain evidence that was adduced as to the effects that allegedly had been brought about in cattle, perhaps from the ingestion of fluorides. Do you, doctor, from research that you have done, know of any evidence to suggest that there is any difficulty in*

reproduction that is brought about by ingestion of fluoride?

Prof. Suttie: *No. Experimental studies have indicated that there is no direct effect of fluoride on the reproductive process.*

Mr. Lamek: *Is there anything in the evidence and the data that would suggest that ingestion of fluoride in any way induces abortion?*

Prof. Suttie: *No, it does not.*

Mr. Lamek: *Or stillbirth?*

Prof. Suttie: *There is not.*

Mr. Lamek: *Or deformity in a calf?*

Prof. Suttie: *There is no evidence to support this.*

Mr. Lamek: *Or underweight calves?*

Prof. Suttie: *There is no evidence, that I know of, to support this.*

583. Professor Willoughby also made reference to the effect of F on reproduction in cattle (Volume VI):

Prof. Willoughby: (p. 966) *If I might, I neglected to mention the influence of fluorosis in animals.*

Chairman: *Yes ...*

Prof. Willoughby: *And in this regard, it has been shown repeatedly that animals that have received an excessive amount of fluoride, and they are actually showing other clinical signs of fluorosis—perhaps some lameness, some weight loss and teeth changes, and so forth—the reproductive tract of these animals continues to function normally. However, there is a secondary involvement in the reproductive system, because obviously if an animal is unable to move normally they would not express visible evidence of estrus or heat, and also an animal that is losing weight ... and it is too painful for the animal to walk around and graze normally. Again, this animal will not likely show evidence of estrus, and will not consequently be able to be bred.*

584. In his review, Cass[50] stated that:

> "The prolonged ingestion of elevated quantities of fluoride has no direct effect upon conception, gestation, or parturition. However, serious dental damage and loss of appetite, with an attendant loss in weight and markedly reduced lactation, may result in the suppression or interruption of estrus, reduction of the weight of newborn, and poor subsequent development of young."

He cites a number of references to support these statements.

585. It thus appears that, while reproductive performance of cattle is not directly affected by the ingestion of relatively high amounts of fluoride over a prolonged period, the indirect effects can nevertheless have important consequences, if the fluorosis is very serious.

586. In connection with swine and poultry, Burns and Allcroft (1966)[62] in their study of "Fluorosis in Cattle," Part I, observed what the general literature has shown, that:

> "Symptoms of fluorosis are not usually found in pigs and poultry kept on affected farms because of the difference in management and feeding between those species and ruminants and because of their shorter economic life span. Pigs were bred and reared over a five-year period and presented no difficulty outside normal pig-keeping experience."

When considering that three Port Maitland farms, where swine were kept, had been declared to be contaminated, the Committee must say that they can find no justification, either in evidence or from the literature, for the payment of $7,500 to those farmers for "unborn piglets due to reproductive problems."

H. FLUORIDE CONTENT OF ANIMAL PRODUCTS

587. Professor Slinger, in his presentation at the hearings, discussed the findings which Largent had reported on the fluoride content of a number of tissues from cattle which had lived for varying periods near a factory in which rock phosphate was being processed. The results are presented in Table XXI[53]:

588. It may be noted from the table that all of the soft tissues and milk were quite low in fluoride. The high content in bone and urine are typical of the situation with fluoritic animals.

589. The fact that the fluoride content of milk, from cows showing definite signs and symptoms of fluorosis, is very low is a very significant one and we wish, in order to assure the public, to cite other studies which prove this point beyond question. Agate and

TABLE XXI

The fluoride content of tissues obtained from three cows that lived on a farm adjacent to a factory in which rock phosphate was being processed.

	Fluoride content (p.p.m.)		
Tissue	F_1*	F_2**	F_3***
Thyroid	—	1.70	—
Stomach	11.0	1.56	—
Intestine:			
Small	1.74	2.74	—
Large	5.16	—	—
Liver	0.80	0.31	0.57
Kidney	4.23	1.30	1.68
Lung	2.82	0.85	1.10
Heart	0.77	0.39	0.43
Brain	3.34	1.35	9.60
Blood	0.68	0.31	0.54
Milk	0.21	—	—
Urine	46.0	12.1	—
Vertebrae	8,000.	14,500.	6,800.
Rib	4,600.	13,300.	6,350.
Metatarsal	9,500.	14,500.	4,950.

*Cow F_1 was placed on the farm at birth and remained there between 8 and 9 years before slaughter.
**Cow F_2 was placed on the farm at birth and remained there 9 years before slaughter.
***Cow F_3 was placed on the farm at 5 years of age and remained there 4 years before slaughter.

his associates, in conducting "The Fort William Study" in 1949, found that the milk from dairy cattle, grazing year-round on fluoride-contaminated pasture land near the aluminum plant, contained 0.11 to 0.44 p.p.m. of fluoride. This is a normal level. In 1955 and 1956, Stoddart and his colleagues at the American Dairy Science Association meetings, reported on their extensive "Fluorosis Studies in Dairy Cows."[63] Their results may be summarized as follows:

> "Lactating dairy cows fed for 40 months on a diet containing 25 p.p.m. F produced milk which had a fluoride concentration of 0.09 to 0.19 p.p.m.; when dairy cattle had been fed for 40 months on a diet containing 50 p.p.m. F the concentration in the milk was 0.13 to 0.21 and when dairy cattle had been fed for 40 months on a diet containing 100 p.p.m. the fluoride content of the milk was 0.13 to 0.35."

In Allcroft, Burns and Herbert's study, "Fluorosis in Cattle" (1965)[52], where the fluorine content of monthly pasture samples over a 7-year period varied from 8-292 p.p.m., and where bone F levels reached up to 7,000 p.p.m.:

> "The mean value of milk for the first three years was 0.45 p.p.m. and for the remainder of the period 0.18 p.p.m. The figure for the latter period was similar to that found in milk in uncontaminated areas."

590. The muscle and visceral meat, from animals suffering from fluoride poisoning, will contain more fluoride than the meat from animals not so afflicted, but the raised fluorine levels, amounting to only 2 to 3 p.p.m. on the dry basis, are still so low as to constitute no hazard to the consumer.

591. The fluoride content of the milk of the dairy cow has been observed to vary with that of the water she is consuming. However, the changes are slight and the fluorine concentrations produced by waters naturally heavily contaminated with fluoride, up to 10 p.p.m., have not been observed to exceed 0.5 p.p.m. Much of the low fluoride concentration in milk is found in the aqueous phase, so that the fluoride content of butter and cheese would be very low. Raising the fluorine content of the cows' ration to relatively high levels does not raise the level of fluoride in milk appreciably.

592. Recent studies with broiler chickens fed a diet containing 34 p.p.m. of fluoride indicated that bone contained 30-50 times the concentration of fluoride found in the meat. Breast meat represented 21 per cent of the carcass minus giblets and contained 0.9 p.p.m. of fluoride, or 1 per cent of the total fluoride in the carcass. Leg and thigh meat represented 25 per cent of the carcass minus giblets and also contained 0.9 p.p.m. of fluoride, or about 1 per cent of the total carcass fluoride.

593. The University of Wisconsin investigators[48] have reported that, when hens were fed a ration containing 1,050 p.p.m. of fluoride from rock phosphate, the fluoride in the yolk increased to an average of 3 p.p.m. from the normal level of 0.8 to 0.9 p.p.m. The yolk was considerably higher in fluoride than the white, which contained only 0.3 p.p.m. on this same diet. Since the albumin represents about 57 per cent of the total weight of the egg, and the yolk only 32 per cent, this would mean that the total edible parts would contain about 1.3 p.p.m. of fluoride, with a diet containing much more fluoride than would likely be encountered in normal feeding practices. In a study conducted by Allcroft

and associates (1965),[52] it was reported that "after about nine months on the farm, the contents and shells of both duck and goose eggs increased to about 3 times the normal values of 0.08 for contents (0.24 p.p.m.)—this increase was not progressive with length of sojourn on the farm." Other workers have reported somewhat higher, and others considerably lower F values, for the contents of eggs. One probable reason for this is the wide variation in dietary calcium levels used. The high calcium levels, fed to laying hens, may well be one reason for the relatively high tolerance of the laying hen to fluoride.

594. Compared with such materials as the tea plant, which will often contain up to 100 p.p.m., and certain sea foods which may contain 5-15 p.p.m., the concentrations of fluoride in meat, milk and eggs are extremely low—even when the animals have received diets containing high levels of fluoride.

595. In this same connection, it is of interest to note that fluoride in soil and water has little or no influence on the fluoride content of edible plant produce consumed by humans. It is common practice to fertilize soil with phosphate fertilizers containing fluoride. Such a practice will increase the fluoride content of the drainage water but the plant fluoride is generally not increased.

I. EFFECT OF SULPHUR DIOXIDE AND ZINC POLLUTION ON ANIMAL PERFORMANCE

596. In the case of humans, working in a contaminated atmosphere in an industrial plant, there is great concern for the toxic substances which may be directly inhaled. In the case of animals, the hazard is not a result of inhaling the polluted air outside the factory, which usually contains a very low concentration of toxic material, but the ingestion of forage which has become contaminated with pollutants from the air.

597. The evidence presented at the hearings (Vol. V, p. 658-677), indicated that sulphur dioxide was being emitted from the Sherbrooke Metallurgical Company Limited plant. This a fairly common air contaminant and can be damaging to vegetation itself, as has been indicated in paragraph 386. It is of interest that, while sulphur dioxide can be present in the air in sufficient quantity to cause severe damage to alfalfa leaves, yet this alfalfa will do no harm to cattle which consume it. This fact was brought out in the experiment related herewith.

598. In a feeding experiment with dairy cattle, Cunningham and others (1937)[64] showed that there was no significant altera-

tion in the feeding value of alfalfa hay when such hay was made from alfalfa subjected to an amount of sulphur dioxide sufficient to cause acute injury to more than 25 per cent of the leaflets. The alfalfa was grown near a smelting works. The trial was a double reversal type, of 90 days duration, and was conducted using 10 lactating cows. The time was divided into three periods of 30 days each. Digestion trials were conducted during the last 10 days of each 30-day period. The alfalfa was fed to the cow at the rate of 1¾ pounds for each 100 pounds of liveweight, along with silage at 1½ pounds per 100 pounds liveweight, and grain at the rate of 1 pound for each 4 pounds of 4 per cent fat-corrected milk. There were no significant differences between the cows, when receiving normal hay or contaminated hay, based on milk production, body weight changes, apparent digestibility of various ration constituents, hay refusal, or percentage of energy ingested which was returned in the milk.

599. It was not surprising that SO_2-damaged alfalfa in the above study was not toxic to cattle. The SO_2 would probably react with the water, in or on the plant, to form sulphurous acid (H_2SO_3), which would in turn be converted to salts such as sodium sulphite (Na_2SO_3), and these compounds would be further oxidized to sulphates such as $NaSO_4$. It is the ability of sulphur dioxide, sulphurous acid, and sulphites, to act as reducing agents which makes them valuable bleaching agents when they come into contact with organic colouring matter.

600. Sulphate can be readily metabolized by cattle (or other animals), and it is common practice at the present time to include sulphur (frequently as sulphate) in rations for ruminant animals, in which much of the protein is being provided by urea. Frequently, 0.50 per cent of $CaSO_4$ is added to the corn silage and the silage fed at the rate of 20 or more pounds per head per day. Some of the sulphur thus fed is converted by rumen microorganisms to form the amino acids methionine and cystine. Much of it is absorbed in the small intestine and is excreted in the urine in proportion to its concentration in the blood serum. The major portion of the amount consumed, which is not required as free sulphate or organic sulphur compounds in the body, is excreted during the first 24 hours.

601. Studies with radio-active sulphur (S35) indicate that sulphate, in free and bound form, is found in almost every tissue and cell in the body and, as such, plays a vital role in the animal.

602. While zinc is a dietary essential for all species of animals, too much of the element is toxic. In recent experiments, conducted

by Ott and co-workers (1966)[65], it was found that zinc levels of 900 p.p.m., provided in the diet (of beef cattle) as zinc oxide, caused reduced weight gains and lowered feed efficiency. Zinc levels of 500 p.p.m. and lower exerted no detrimental effects. Zinc levels of 1,700 p.p.m. and higher caused reduced feed consumption and depraved appetite, characterized by excessive consumption of salt and other minerals, as well as wood chewing. These same workers investigated the toxicity of zinc on lambs[66] and reported that zinc consumption above 1,500 p.p.m. of diet caused depressed feed consumption, while 1,000 p.p.m. caused reduced gains, decreased feed efficiency, and increased mineral consumption. Prolonged consumption of toxic levels of zinc caused death.

603. The symptoms of reduced weight gains, lowered feed intake, and inefficient utilization of feed, have been reported for several species of animals and appear to be general findings. Pigs and poultry appear to tolerate zinc to a somewhat greater extent than ruminant animals, with levels of 1,800-2,000 p.p.m. being required to cause depressions in growth rate and feed intake. Because pigs and poultry are not normally kept out of doors on pasture, and since they must be fed mainly on concentrate feeds for satisfactory economic returns, it would be very unlikely that these species would be affected by zinc emitted into the atmosphere.

604. According to the Agricultural Research Council publication, *The Nutrient Requirements of Farm Livestock, No. 2, Ruminants* (1965),[67] "there are no reports of zinc toxicity in ruminants, but it has been found that the retention of calcium and phosphorus by sheep was significantly reduced by the addition of 0.5 or 1.0 per cent zinc sulphate to a basal diet." While we know that zinc sulphate was present in the samples taken from the light bulbs and from the car, as exhibits presented at the hearings (Appendix III), we have no figures on the amount present in forage crops, the latter being the largest component of a ruminant's diet. It is of interest that the above-quoted report suggests a dietary intake of 50 mg. zinc per kg. feed, on a dry basis, for ruminants as a nutritional standard.

J. GENERAL DISCUSSION

605. In the body of the report, a review of the relevant literature has been presented. Consultants to the Committee gave evidence regarding the effects of the ingestion of higher-than-normal levels of fluorides over long periods of time. Such evidence has also been included in the report. There was general agreement between

the consultants and other scientists as to the validity of the conclusions on this aspect of the subject.

606. The condition of fluorosis has been discussed in some detail and the criteria for the adequate diagnosis of the condition have been considered and specifically stated. Some of the signs and symptoms, which might superficially resemble some of the systemic symptoms of fluorosis, have been presented. Thus, after differential diagnoses have been considered, it is possible, when the specific diagnostic criteria are used, to establish, without question, the diagnosis of fluorosis.

607. It has been shown, in the body of the report, that the ingestion of fluoride in abnormally high concentrations does not result in high fluoride levels in the soft tissues of the animal—even in an animal suffering from fluorosis. Thus it was established—and this has been known for many years—that the ingestion of high levels of fluoride in the fodder or water has no significant effect on the meat or milk of the animal.

608. We have presented evidence from the literature, from the evidence of the Committee's consultants, and from the evidence of other experts, taken at the hearings, to show that the ingestion of fluorides, even at levels which produce definite clinical fluorosis, does not have any direct effect on the reproductive system of animals, and that it does not, to be specific, have any direct effect on oestrus, coming into heat, conception, future gestation, birth of crippled or malformed calves, or on reproduction in general. It has been shown, too, that any decreases in fertility, or other reproductive difficulties, are secondary manifestations of the physical and nutritional condition of the animal.

609. The condition of fluorosis in cattle is not *per se* an indication of economic or productive loss or damage. Evidence given at the hearings, by Dr. Suttie and Dr. Nelson, confirm this point. Other evidence, too, substantiates the fact that cows which have been "diagnosed" as having fluorosis have been retained in the herds and have continued to reproduce well. It has likewise been brought out in evidence that cows, which have had fluorosis to the extent that lameness was apparent, have continued to produce and have recovered from their lameness, after appropriate corrective changes in management were instituted.

610. It has been brought out in evidence that, on adjacent farms, one farmer may have "experienced" fluorosis in some of his cattle, while his neighbour had no "problem" whatsoever. We can find no justifiable reason to "condemn" a herd simply because

one or two or even 10 cattle in a herd of 40 or 50 have varying degrees of fluorosis. The loss of productive capacity is a very significant and realistic criterion when determining the effects of fluoride from a practical point of view.

611. A great deal of information is available on the fluoride concentration of the urine, in relation to the so-called lifetime exposure to fluoride ingestion. Although up to 10 p.p.m. of fluoride in the urine is considered to be within the normal range, it is not correct, we feel, to conclude that levels above 10 p.p.m. are necessarily indicative of economic damage. Indeed, even at a level of 20 p.p.m., other and more conclusive evidence of fluorosis—such as lameness, dental defects, etc.—should be available before a firm diagnosis can be made and economic damage established.

612. Evidence was brought forth which established the fact that some cattle, which had been lame, had indeed improved in condition and in productivity and that the lameness had disappeared. This improvement in general condition of some cattle can be related to the "limed candle" result, which indicated that the extent and concentrations of fluoride-containing emissions from ERCO had, in general, decreased in recent years, subsequent to the rapid increase beginning in December, 1964, and continuing through 1965.

613. There are few sheep raised in the townships of Dunn, Sherbrooke, and Moulton—how many, of course, is not known to the Committee. As far as the evidence is concerned, Dr. Nelson stated that he had examined sheep on the Niece brothers' farm and found evidence of both bone lesions and dental lesions which, according to his evidence, were caused by fluorides.

614. He diagnosed the condition as "fluorosis" but, unfortunately, from the Committee's point of view, did not confirm his diagnosis with supporting chemical analysis of urine or bone, or both. In addition, the Committee was not satisfied that other diseases of sheep, which result in clinical signs indistinguishable from those found in fluorosis of sheep, were eliminated in the differential diagnosis of fluorosis on the sheep inspected. The Committee is willing to accept the diagnosis, in consideration of Dr. Nelson's opinion, along with indisputable evidence of fluorosis in an equally susceptible species, i.e. cattle raised on adjacent farms.

615. The literature relative to the effects of fluorides on hogs has been presented in the body of this report, and it is accepted that hogs are considerably less susceptible to fluoride ingestion than are cattle and sheep. On the basis of evidence by the Committee's

consultants, and knowing the concentrations of fluoride in samples of cereal grains in the area, it is considered most unlikely that fluorides were responsible for any of the losses or damage in hogs experienced by farmers in the area. There is no evidence that the teeth of the hogs were examined, that urine samples were taken, that bone exostosis was present, or that chemical analyses of bone were conducted. In fact, two of the area farmers who had problems with their hogs did not even suggest that fluorides were responsible for the conditions of their swine. Veterinarians in the area did not diagnose fluorosis in any of the hogs, in spite of Dr. Nelson's feeling that the sows on one of the farms were affected. In the light of no testing at all by them, and contrary evidence as obtained from the literature, from Professor Crampton, and from the Committee's consultants, the Committee finds that there is no evidence to support the contention that fluorides have had any deleterious effect upon the area hogs.

616. In at least one instance, the hogs were reported to be overly fat, and the farmer purchased no commercial supplement to balance out his home-grown grain. This could well mean that his problem in reproductive performance was purely a nutritional consideration. Further evidence that fluoride toxicosis was not involved is that the sows in question apparently showed none of the symptoms attributable to poisoning with this element, e.g. lameness, emaciation, etc.

617. There are a great number of nutritional and infectious conditions which affect poultry and which produce marked bone and other skeletal changes, including joint swellings and deformity. Again, it is well known among nutritionists that poultry are extremely resistant to fluorides, and indeed experiments have been conducted in an attempt to find out the reason for the high resistance of poultry to fluorides. It is known, too, that, although the eggshell of eggs laid by poultry on a high fluoride intake contains higher than normal amounts of fluoride, the whites and yolks contain normal amounts of fluoride.

618. We include bees under the category of livestock, simply as a matter of convenience. Evidence, from one of our consultants on the basis of an extensive review of the literature, indicates that bees are generally susceptible to higher than normal concentrations of fluorides, but that the toxicity depends upon several factors. Since only inadequate data are available—no dead bees were analysed for fluoride content—we can but hazard a guess that damage to bees in the area has resulted from higher-than-normal levels

of fluorides in the atmosphere and particularly on the flowers of growing vegetation.

619. Experimental feeding studies with animals have been conducted, using single sources of fluoride such as sodium fluoride, calcium fluoride, fluoroapatite. The fluoride in sodium fluoride is about twice as readily "available" as that in calcium fluoride or fluoroapatite. While most of the fluoride emitted from phosphate plants would be expected to have the same order of "availability" as sodium fluoride, analytical data are not available as to the relative concentrations of different forms of fluoride actually being emitted from the ERCO plant. It is therefore difficult to relate fluoride concentrations, in forage and other crops being fed to area livestock, to experimental results reported in the literature.

620. The available evidence suggests that domestic animals, which consume crops contaminated with reasonable levels of sulphur dioxide, are not harmed thereby. The tolerance of animals to zinc is fairly high; however, in the absence of data on the extent of contamination with zinc of forage in the area, no definite statement can be made with respect to the possibility of zinc pollution being deleterious to animals grazing in the area or consuming forages harvested and fed during the winter. Nor can a definite statement be made concerning the possibility that there might be interactions between fluoride, sulphur dioxide, and zinc, if forages contaminated with all three are consumed. The Committee was disturbed that the Sherbrooke Metallurgical Company showed a complete lack of concern about its emission "problems."

621. The Committee appreciates fully that apprehension and discouragement developed in the minds of many persons, not only from the human health point of view but relative to their cattle. To see lameness develop in a cow, to see her teeth become progressively damaged, to witness progressive loss in her condition, and to see her refuse to forage in "good pasture," naturally aroused concern that the "unknown" was devastating their herds and challenging their respected way of life. It tended to arouse suspicion, mistrust and over-anxiety and, as a result, many unjustified claims were made—not necessarily in a spirit of dishonesty, but in light of the unknown—almost in the spirit of despair. Good communications could have prevented most of the emotional upheaval.

622. "Hind-sight is better than foresight." In our general conclusions, comments will be made about the system of awards and the method of awarding compensation. It is not the responsibility of this Committee, however, to be judge of specific claims.

623. That ERCO agreed to pay compensation for losses and damages to livestock, crops, etc., admits a definite responsibility, at least for such losses. On the other hand it does not, in the opinion of the Committee, warrant the assumption that all losses and damages to cattle and production over the pertinent years is their responsibility. There is no question that ERCO has paid more for losses and damage to farm livestock than can be justified on the evidence available. They appear to have tried to make up for inadequate control of emissions by over-compensation in the name of public relations.

K. SUMMARY OF EFFECTS OF POLLUTANTS ON ANIMAL HEALTH

624. In order to diagnose fluorosis accurately, as well as to assess the degree of severity of the condition of animals, we list again the following criteria, which have been established by expert consultants and which reflect accepted data from the scientific literature:

(a) the fluoride content—

 (i) of the food ingested

 (ii) in the bones or teeth

 (iii) of the excreted urine.

(b) the degree of mottling and staining of the teeth, the rate of damage of teeth, of hypoplasia of the enamel and excessive wearing of the teeth.

(c) systemic evidence as indicated by lameness, anorexia, inanition, cachexia, exostosis and bone changes.

625. The evidence presented to the Committee substantiates the claim and observations in the area that:

(a) Mild (Grade I) fluorosis did occur in some dairy cattle.

(b) damage due to fluorosis (Grade IA, no lameness) did occur in other dairy cattle.

(c) Grade II fluorosis (herds slightly affected with lameness, i.e. only a few lame animals) did occur in some of the dairy herds.

(d) Grade III fluorosis (10-30 per cent of the herd lame, with urinary values of 20-60 p.p.m. F) did occur in at least two herds.

(e) Grade IV fluorosis (urinary values of 60-80 p.p.m. F) was observed in a few animals. Lameness, gross abnormalities, loss of condition and secondary loss of weight and decline in milk production, were observed in these few cases.

626. There was no evidence that any cattle died of fluorosis, although a few were in such poor nutritional health that they had to be destroyed.

627. There was no evidence that any of the cattle's teeth "fell out."

628. Although some herds and some "premises" were described as contaminated, only a few cattle in these herds showed evidence of fluorosis.

629. Several herds in the "cone" area showed no evidence of fluorosis of any degree, and many cattle in the "contaminated" herds did not show any evidence of fluorosis.

630. Although a level of 10 p.p.m. of fluoride in animal urine is considered to be within a normal range, it is not correct, we feel, to conclude that levels above 10 p.p.m. are necessarily indicative of economic damage. Some cattle did not have high urinary values (20 p.p.m. or more) nor did they show any symptoms associated with fluorosis—lameness, loss of condition, loss of body weight, and loss of milk production—yet full compensation was paid for such animals. In some cases, farmers retained cattle for which they had received compensation, and these cattle continued to be "producing" animals; in other cases, cattle were sold and the sale price was retained by the farmer, in addition to the compensation already paid by ERCO. Even when urinary values exceed 20 p.p.m. F, other and more conclusive evidence of fluorosis—lameness, dental defects, etc.—should be ascertained before a firm diagnosis can be made and economic damage established. Some cases of "lame cattle" were diagnosed by qualified veterinarians as laminitis (a condition similar to fluorosis in its crippling aspects). In spite of the similarities of these two disorders, no consideration was given to the possibility of examining area animals for the further incidence of laminitis, which is a common disorder of cattle.

631. Evidence from the scientific literature, and the evidence presented by the Committee's consultants and other experts, showed that the ingestion of fluorides, even at levels which produce clinical fluorosis, does not have any direct effect on the reproductive system of animals. In addition, such ingestion does not have any direct effect on oestrus, conception, fertility, gestation, birth of mal-

formed calves or on reproduction in general. Any decrease in fertility, or other reproductive abnormalities, can only be associated with extreme debility of the animal. In spite of these facts, several farmers presented claims, on the basis of decreased fertility of their animals, and some of these claims were honoured.

632. Some cows and some herds suffered a loss in milk production, for which compensation was paid. It should be emphasized, however, that this loss in production was not a primary result of fluoride ingestion, but was a secondary factor related to a failing appetite and a general loss of weight. Even so, it is fully appreciated that, to the farmer, the distinction between a primary result and a secondary factor is meaningless.

633. It was brought out in evidence at the hearings that when some changes were made in farm management, feeding practices or type of herd carried, productive and profitable farming was possible in the area even under the conditions of the past few years.

634. Swine and poultry are considerably less susceptible than cattle, yet evidence showed that compensation was paid for alleged damage to both classes of livestock. Although it was indicated in the evidence that fluorosis was found in some sheep, it is difficult to accept this evidence as valid. No evidence was presented at the hearings which showed that, under reasonably good farm practice, swine were adversely affected. The claims that reproduction problems, including reduced litter size and failure to conceive, were caused or influenced by fluorides could not be accepted on the basis of the literature and on the basis of other evidence given at the hearings. It is suggested that other reasons and other causes must be sought to account for such a situation. This could well be a problem for future experimental study.

635. Knowing that dairy cattle are 5 to 6 times as susceptible to high concentrations of fluoride as are chickens and turkeys, and since a small proportion only of the cattle were adversely affected by the fluoride concentration in the area, it is difficulty, certainly with our present knowledge, to accept as valid, claims that poultry in the area were affected by fluorides. Here again, further research and experimentation might be carried out to determine why poultry are so resistant to fluoride and whether flocks raised under contaminated range conditions could indeed develop fluorosis.

636. There was evidence presented which supports the contention that bees may have been affected by the fluoride concentration in the area.

637. No direct evidence was brought forward nor did the Committee initiate any studies on the effects of fluorides on wild life. It was reported in evidence that song birds seemed to be scarce in the area.

638. In connection with the evidence dealing with livestock in general, it was apparent that there was lack of communication between the various agencies, persons and farmers from whose cows or herds samples of urine, etc., had been taken.

639. There has been an air pollution problem in the area, resulting in loss and damage from fluoride emissions, which has existed for several years, yet there appeared to be an unacceptable lack of knowledge, on the part of many, about fluorosis and the necessary criteria on which a diagnosis must be made.

CHAPTER VI

An Economic Analysis of Changes in the Agriculture of Dunn, Moulton, and Sherbrooke Townships, 1956-66

640. The main source of statistical information, the *Canada Census,* does not provide sufficient detail to develop a precise picture of the changes in the agriculture of the three-township area over the past 10 years. A detailed analysis would be possible only if complete farm business records were available from a large percentage of farms in the area in question; such records are not normally available. However, the information available does allow a general analysis which reveals some important facts relative to the economic structure of agriculture in the area.

641. A commercial farm is defined in the *Canada Census* as a farm with gross sales of at least $2,500 during the year preceding the census enumeration. Farmers who obtain less than $2,500 in gross sales from the farm usually either have small farms or are part-time farmers, and nearly all must rely on off-farm sources of income to provide an adequate income level. Such farms may be classified as non-commercial farms.

642. The three-township area had a significantly lower percentage (40) of commercial farms than the rest of Haldimand County (56) in 1961. The same was true in 1966, with 51 per cent commercial farms in the three townships and 66 per cent in the rest of Haldimand County. Two points are significant: The percentage of commercial farms increased between 1961 and 1966 in the area, as rapidly as in the rest of Haldimand County, as would be ex-

TABLE XXII

CHANGE IN NUMBERS OF FARMS AND FARM SIZE

	Moulton, Dunn and Sherbrooke		*Rest of Haldimand Townships*	
	1961	*1966*	*1961*	*1966*
Number of commercial farms	147	159	958	1,067
Number of non-commercial farms	218	155	747	556
Commercial farms as % of total farms	40	51	56	66

pected if no serious upsetting force was operative. Second, the percentage of non-commercial farms, e.g. part-time farmers, was substantially higher in this area than in the remainder of the county, as might be expected in an area with greater soil-productivity problems coupled with a convenient source of off-farm employment.

TABLE XXIII

THE CHANGE IN GROSS SALES FROM AGRICULTURAL PRODUCTS BETWEEN 1960 AND 1965

Percentage Increase 1965 Relative to 1960 in:	*Moulton, Dunn & Sherbrooke*	*Other Haldimand Townships*
Total Gross Sales from all farms	+38.5	+35.5
Average Gross Sales from commercial farms	+38.7	+27.7

643. The area showed an increase in gross sales from farm products similar to, and in fact, slightly above the increase shown in the rest of Haldimand County during the 1960 to 1965 period, the period for which data are available. Commercial farms accounted for 86 per cent of the gross farm sales from the three township areas in 1960, and 94 per cent in 1965. Gross sales from the commercial farms in this area not only rose as rapidly as in the rest of Haldimand County, between 1960 and 1965, but at a substantially higher rate, e.g. 38.7 per cent versus 27.7 per cent. The gross sales picture cannot be interpreted to mean that the commercial farms in this area had net farm incomes rising at a faster rate than in the rest of the county; it is likely that net incomes rose at least as fast, but the information is not adequate to determine

the exact net income picture. Hogs, poultry and beef finishing increased more, and dairy cattle decreased more, in this area, relative to the rest of the county, during the 1960-65 period. Ontario farm business records indicate that normally poultry, hog, and beef finishing enterprises must show at least 10 per cent more gross sales than dairy operations to provide the same net income.

644. The total acreage used for crop production in Haldimand County, excluding the three townships, was approximately the same in 1966 as in 1956, although there was an increase of 1.7 per cent between 1956 and 1961, and a decrease of 2.3 per cent between 1961 and 1966. An important difference occurred in the three township area, in that there was a reduction of approximately 15 per cent in acreage used for crop production between 1956 and 1966. It is important, as well, to note that the decline between 1956 and 1961, at 8.8 per cent, was at least as great as that (6.6 per cent) between 1961 and 1966. These acreage figures do not include the acreage used for unimproved pasture and, of course, some of the acreage shift, if moved into this use, could contribute significantly to the production of dairy and beef cattle (Table XXIV-A).

645. Study of the changes in acreages of individual crops reveals that the total acreage of fruits and vegetables in the three townships declined each intercensal period by more than 100 acres. In the rest of the county, the same change occurred between 1956 and 1961, but then the acreage held steady during the 1961 to 1966 period. The vegetable acreage in the three townships dropped sharply in the 1956 to 1961 period, and again in the succeeding period, but the drop was not as sharp; potato acreage has held relatively steady; tree fruit acreage was variable; and small fruit acreage declined markedly.

646. The grain crop acreage has shown little change in total in the three townships in the years 1956 through 1966. One point should be noted: The acreage of feed grains has increased to offset the decrease in the acreage of winter wheat, which is marketed mainly as a cash crop. Forage crop acreage has declined significantly in the area, while in the rest of the county the acreage has held relatively steady.

647. The numbers of animals and poultry have been converted to a common animal unit, in order to study the patterns over the 10-year period (Table XXIV-B). The total intensity of livestock and poultry production increased in the three-township area, as it did in the rest of Haldimand County. The important point here is the composition of the livestock and poultry population. There

TABLE XXIV—A

CHANGES IN ACREAGE OF FRUIT, VEGETABLE, AND FIELD CROPS, 1956-1966

	Moulton, Sherbrooke and Dunn			*Other Haldimand Townships*		
	1956	*1961*	*1966*	*1956*	*1961*	*1966*
Acreage of Fruit, Vegetable, and Field Crops						
Fruit and Vegetable Crops						
Vegetables	495	297	242	798	730	718
Potatoes	41	66	47	45	40	33
Tree Fruits	84	169	68	92	52	67
Small Fruits	56	34	16	29	18	29
Total	676	566	373	964	840	847
Grain Crops						
Grain Corn	3,102	2,348	3,647	3,596	1,305	6,601
Oats	1,870	3,156	2,866	31,205	41,291	36,432
Winter Wheat	4,154	3,859	1,538	21,797	18,945	7,125
Barley	76	123	1,058	646	1,046	4,533
Mixed Grain, Rye, Flax	337	222	362	4,353	2,325	5,200
Total	9,539	9,708	9,471	61,597	64,912	59,891
Forage Crops						
Hay	9,963	8,159	7,467	66,083	65,428	73,151
Improved Pasture	6,385	5,913	5,253	45,044	46,115	37,996
Corn Silage	930	764	916	4,686	4,577	6,784
Total	17,278	14,836	13,636	115,813	116,120	117,931
Other Field Crops	197	144	126	286	130	487
Total Acreage in Crops	27,690	25,254	23,606	178,660	182,002	179,156
Change in Total Acreage in Crops		—8.8%	—6.6%		+1.7%	—2.3%

TABLE XXIV—B

CHANGES IN NUMBERS OF LIVESTOCK AND POULTRY*, 1956-1966

	Moulton, Sherbrooke and Dunn			*Other Haldimand Townships*		
	1956	*1961*	*1966*	*1956*	*1961*	*1966*
Forage Consuming Animal Units**						
Dairy Cows and Heifers	2,947	2,944	2,447	21,416	25,576	24,523
Beef Cows and Heifers	411	604	589	3,401	3,939	4,808
Steers	200	371	436	2,288	2,420	3,216
Bulls	90	77	60	530	500	495
Sheep	67	46	62	565	544	563
Horses	286	149	165	1,112	758	740
Total	4,001	4,191	3,759	29,312	33,737	34,345
Grain Consuming Animal Units***	735	809	1,433	4,368	4,652	6,575
Hogs	474	509	814	2,445	2,331	2,618
Chickens	1,209	1,318	2,247	6,813	6,983	9,193
Total						
Total Animal Units	5,210	5,509	6,006	36,125	40,720	43,538

*The numbers are in terms of animal units, with 1 unit equalling 1 dairy cow, or 2.5 hogs over 6 months, or 5 hogs under 6 months, or 100 chickens over 6 months, or 238 chickens under 6 months, or 7 sheep.

**Hay, pasture, and silage form the basis of the ration for these animals.

***Grains form the basis of the ration, with little or no forage consumed.

is greater fluorosis danger to the animals which consume forage; dairy and beef cattle are the important ones from a farm income standpoint. Dairy cattle numbers were not following the same trend in the three townships, between 1956 and 1961, as was the case in the rest of the county; the numbers held steady in the area but increased in the rest of the county. Also, during the 1961-1966 intercensal period, the dairy cattle numbers dropped sharply—a significantly sharper decline than occurred in the rest of the county. Beef cattle numbers showed different trends as well. Between 1956 and 1961, a much larger increase occurred in the area than expected, based on the trend in the county. This level held and increased slightly during the 1961-1966 period.

648. Hogs and poultry, which are least susceptible to fluoride injury, increased in importance in the three-township area, moderately between 1956 and 1961, and markedly during the 1961 to 1966 intercensal period.

649. The change in investment in livestock, machinery, equipment, land, and buildings per area is as follows:

TABLE XXV

CHANGES IN FARM INVESTMENT, 1961-66

	1961 Investment	*1966 Investment*	*Change 1961-66*
Moulton, Dunn and Sherbrooke	$24,265	$36,479	+50
Rest of Haldimand Townships	$29,092	$38,649	+33

650. The significant point is that, although this area was following the same trend as the rest of the county, the rate of increase in farm investment was even higher. Probably the reason for the greater percentage increase is that the base for the area included a higher percentage of non-commercial farms, hence the area was susceptible to change.

651. The number of rural properties that have been sold increased throughout the area during the 1956 through 1966 period, although the rate of increase dropped off a little in the past three years (Table XXVI). Moulton and Sherbrooke had similar rates of increase in numbers of properties sold; Dunn showed a lower rate of increase.

652. It is significant to note that property values have risen on the average, in the three-township area, as evidenced by the in-

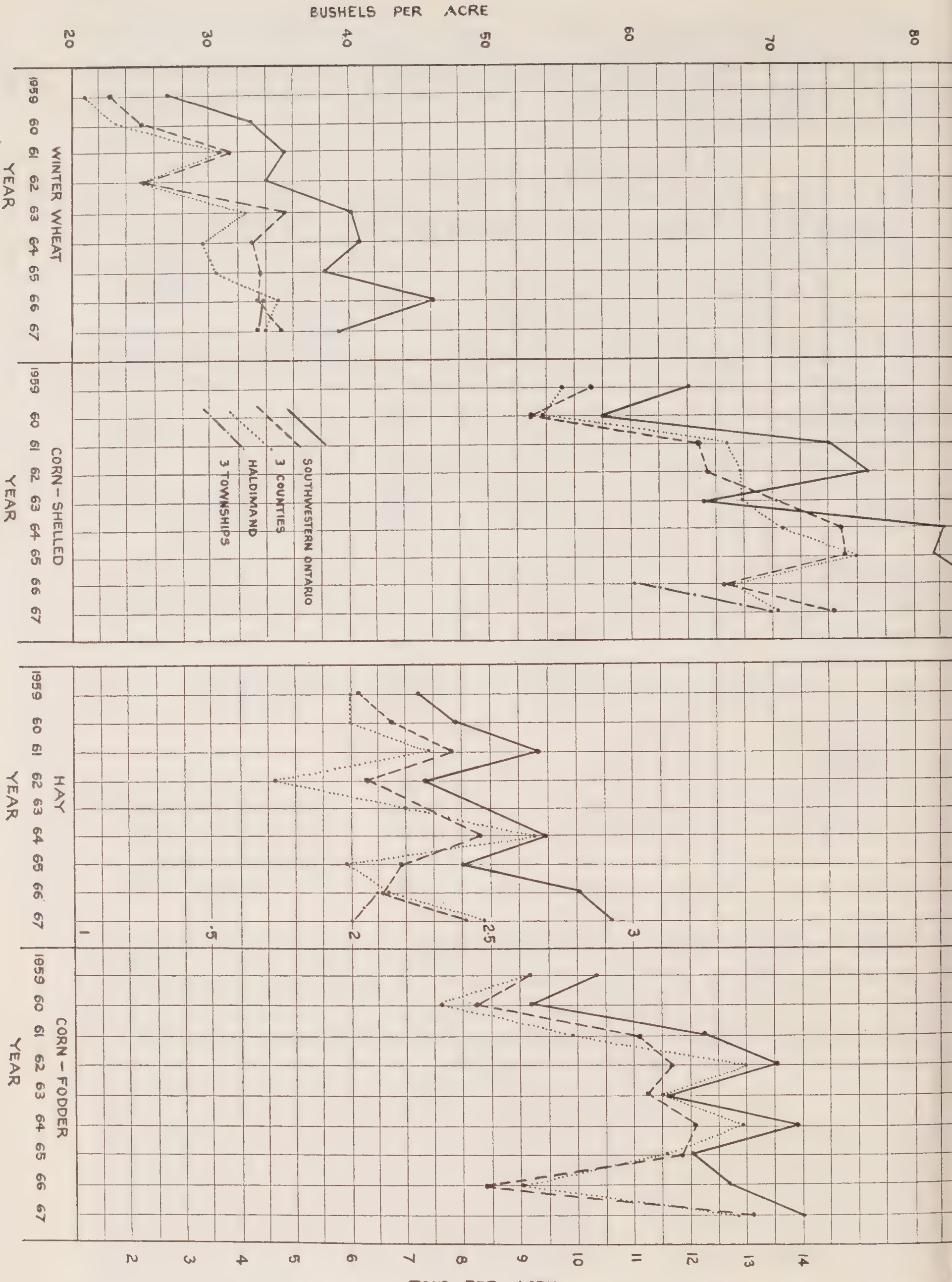
crease in percentage of property sales in the above-$9,600 category. During the 1962 through 1967 period, the percentage increase in above-$9,600 property sales was 7, 11, and 3 per cent for Moulton, Dunn, and Sherbrooke, respectively.

TABLE XXVI

SALES OF FARMS AND OTHER RURAL PROPERTIES

	Moulton		*Dunn*		*Sherbrooke*		*Total 3 Twps.*	
	No.	*% of Total*	*No.*	*% of Total*	*No.*	*% of Total*	*No.*	*% of Total*
1956-1958								
Under $9,600	77	82	66	76	25	83	165	80
Over $9,600	17	18	20	24	5	17	42	20
Total:	94		83		30		207	
1959-1961								
Under $9,600	78	66	74	84	40	75	192	74
Over $9,600	41	34	14	16	13	25	68	28
Total:	119		88		53		260	
1965-1967								
Under $9,600	105	59	76	72	50	81	231	67
Over $9,600	74	41	29		12	19	115	33
Total:	179		105		62		346	

653. In conclusion, it may be said that, although the three Haldimand Townships of Dunn, Moulton, and Sherbrooke lie within a favoured climatic region with respect to agricultural activity, these townshps have not achieved comparable productivity in agriculture when compared with the neighbouring counties in southern Ontario. There are fewer large-scale farming operations in these three townships, total acreages of individual crops have decreased more rapidly than in neighbouring areas, farm investment is lower, and the area has a higher percentage of non-commercial farms, with more off-farm employment required to supplement a smaller farm-based income (see also Figure 7).



Comparison of Crop Yields of Winter Wheat, Corn-Shelled, Hay, and Corn-Fodder, for Years 1959 through 1967 — 12 Southwestern Counties — 3 Counties: Haldimand, Welland, Wentworth — Haldimand County separately — 3 Townships: Moulton, Dunn, Sherbrooke.

Fig. 7

CHAPTER VII

Other Damage—Physical

654. Besides direct damage to animals and vegetation, and indirect loss of trade through psychological influences, there was evidence which showed that direct physical damage has been caused by the emissions from the ERCO and/or Sherbrooke Metallurgical plants. This type of damage may be considered as a particulate contact damage of inanimate materials such as glass, metal, paint, etc. This type of damage, together with a dust deposit nuisance, is brought out in the following evidence from Volume X.

(a) MR. G. DICKOUT

Mr. G. Dickout lives in Stromness Village, less than 1 mile from the ERCO plant lagoons and NE of them. He brought with him a pane of window glass removed from a backyard shed on his property.

Mr. Dickout: *(p. 1515)* *In fact, you can wash it and it will stay like that* [frosted appearance]. *You can see where the putty has stuck to it—it is clear and you can feel the two sides of it and can easily tell which is the outside. The windows in my home are the same way, only not to that extent.*

Mr. Gordon: *Have you tried washing the windows in the house?*

Mr. Dickout: *Yes, I have washed them with vinegar even.*

Mr. Gordon: *What happened?*

Mr. Dickout: *Nothing; when that is damp, whether it is water, vinegar, or whatever you use, it is clear, but the minute it dries it goes back to that condition.*

Mr. Gordon: *Are all the windows in your house similar to the sample of glass you have brought here today?*

Mr. Dickout: *Yes.*

Mr. Gordon: *It doesn't make any difference whether they are on the north side of the house, the east or the west?*

Mr. Dickout: *(p. 1517)* *Yes, the westward side I would say, is possibly worse.* [The prevailing winds blow from the SW, see Figure 1.]

Mr. Dickout: *As I was going to get my car to come over here this morning, I have a Volkswagen, which has not been driven for the past two weeks, sitting in my garage. The garage door has been open most of the time, let us put it that way, and it has been covered with this white dust since last week some day. It arrived overnight.* [The Commissioners have had this dust analysed by x-ray diffraction. It is mostly gypsum, indicating that it came from ERCO ponds. There would be some fluoride associated with the gypsum (see Appendix III).]

Mr. Dickout: *So I took a brush and I brushed off some of this dust and put it in this envelope and I would like to give that to you, too.*

Mr. Gordon: *Do you know what that dust is?*

Mr. Dickout: *I don't know for sure, but we have that occasionally, more so in the winter, when this lagoon, where their* [waste products] *are dumped, freezes over and it is not covered with water.*

Mr. Gordon: *Do you know whether it would be gypsum from the plant which we have heard about in that area?*

Mr. Dickout: *I could not say, but I know this, if it lays on chrome or something like that ... I have a boat in there, too, which I have to keep covered up all the time ... it will rust over in a short period of time.*

Mr. Gordon: *How long has this dust similar to what you have filed in this envelope been coming onto your property, do you know?*

Mr. Dickout: *Oh, I would say 1963. Sometimes you can go out and walk through it on your steps—not this year, but other years. When you walk on your steps you will even leave your imprints or tracks right on it. You can sweep it up with a broom.*

Mr. Gordon: *(p. 1519)* *At all seasons of the year, or just one season in particular?*

Mr. Dickout: *More so in the winter.*

(b) MRS. J. MacKEIGAN

Mrs. J. MacKeigan lives 2 miles NE of Port Maitland. She gave comparable evidence regarding the dust and general nuisance effects of the plants.

Mr. Brooks: *(p. 266)* *From your own health standpoint, have you been conscious of dust or fumes in and about your household?*

Mrs. MacKeigan: *Yes.*

Mr. Brooks: *How long has that been in effect.*

Mrs. MacKeigan: *Well, our house is a new house, built in 1964, and our front window—we don't even clean it because it can't come clean. Something pits right into the glass.*

(c) MR. W. SIDDALL

Mr. W. Siddall lives ½ mile W of the plant area. He operates commercial fisheries in and around Port Maitland.

Mr. Siddall: *(p. 1526)* *I have brought two bulbs here. This bulb was taken where there is a cornice of four feet and it has a two-foot protection out of the weather. That bulb has not been in much more than six months.* [This light bulb was slightly "frosted".]

Mr. Gordon: *Where was this bulb taken from?* [A second bulb was also presented.]

Mr. Siddall: *It was off the corner of the residence cornice and it was under the cornice at least one foot.* [This bulb was heavily "frosted" and had been in place about six or seven months.]

Mr. Gordon: *When did you first notice any of your outside light bulbs were being affected?*

Mr. Siddall: *About 1963.* [As with the dust from the car, x-ray diffraction examination indicated considerable gypsum. There was also some lead sulphate on the bulbs plus some zinc sulphide (see Appendix III).]

Mr. Gordon: *Were you concerned about this condition?*

Mr. Siddall: *Well, yes, we were most concerned, but you let things go on. I contacted Mr. Drowley along with other citizens of the area. Mr. Drowley visited our premises. He told us there was a penetration into the glass and the windows. I said, "We can't get them clean." He said, "You will never get them clean, there is a penetration there." The boat windows, they fog up. They are penerated along with the building. Now the brass fittings, such as horns, they are highly polished and now they are almost green and you are unable to polish them. It is impossible. The cadmium fittings on the boat, two to three weeks—well, they are just like a turnbuckle. You couldn't loosen a turnbuckle when it was* [exposed for] *approximately two weeks. The window frames in the boats, some of them are aluminum, and they are all tarnished and it is impossible to polish them or clean them.*

Mr. Gordon: *In connection with your boating operation, you would have to replace the* [window] *glass periodically, wouldn't you?*

Mr. Siddall: *Yes.*

Mr. Gordon: *Otherwise, it would constitute a navigational hazard?*

Mr. Siddall: *That is right. I contacted the ERCO plant on many occasions and they sent representatives around, they investigated and advised that we had troubles, but then they never came back.*

(d) MR. GEORGE MARTIN

Mr. George Martin lives ½ mile S of Port Maitland. Mr. Martin is employed by Stelco in Hamilton, lives in Port Maitland and operates a "hot dog" stand during the summer season.

Mr. Gordon: (p. 1534)	*What is it you wanted to tell the Commission?*
Mr. Martin:	*Well, since I have been there I had to paint in 1965 and I had to paint in 1966 and I will have to paint again this year. I have replaced the glass, most of the glass once. It needs replacing again. Almost any morning you get up you will find a fallout on your cars and we use outside tables there, and it is just impossible to keep them clean.*
Mr. Gordon:	*Do you notice whether this condition is prevalent all the time, or just at certain times of the year?*
Mr. Martin:	*I would say, yes.*
Mr. Gordon: (p. 1536)	*Have you noticed whether your business decreased last year or not?*
Mr. Martin:	*Well, I can't say that, but there are times when we just have to close the business up and get in the car and leave the area altogether, and this is affecting most of the people up the road. They will all have to get out and leave their places.*

655. Other persons, while giving evidence on other facets of the problem, mentioned, either in passing or incidentally in cross-examination, the dust factor, the odour from the "dust" and "fumes" on certain days, and the effects of the "pollutant" on glass, on paint, on fencing, etc. These persons either lived in the immediate vicinity of the plants or in the direct line of the prevailing winds, usually within a distance of ½ mile from the plant. There is no question that loss and damage has occurred as a result of the emissions, whether from the plants proper or from the lagoons, coming in contact with inanimate objects.

656. The evidence also shows that ERCO recognized a responsibility in this connection (Volume IV, p. 529) when Mr. Pepper, Counsel for ERCO, stated:

"There have, in addition to the complaints about cattle and about crops, I admit, been other complaints, and in examining the material I think you will find to some extent they are justi-

> fied, too. We don't try to hide them. One of them concerns dust. This has been a problem, it has been a sporadic problem. It depends on climatic conditions. Another is the effect of fluoride on glass in the immediate vicinity of the plant. Within a short radius of the plant, ERCO accepts a responsibility and instructs me that it intends in the spring of this year to replace the glass of anybody who can persuade us he has been seriously affected."

657. Although ERCO has belately demonstrated its willingness to pay for the replacement of damaged glass, the Committee must state again that such concern was too late, and that much expense and many difficulties could have been avoided had ERCO foreseen probable damage and instituted adequate preventive measures at the proper time.

CHAPTER VIII

Summary of the Activities of the Ontario Water Resources Commission in the Port Maitland Area

658. Since the Ontario Water Resources Commission (OWRC) has been involved in the control of water pollution in the Port Maitland area for some years, a very brief résumé of its purposes and function is herewith presented. Established by legislation in 1957, the OWRC is responsible for the development, utilization, treatment, and management of water resources, including the provision of adequate water pollution control measures throughout the province.

659. The Division of Industrial Wastes deals directly with industry in all problems which arise from the need of industry to dispose of wastes resulting from various manufacturing processes. The contents of most of these wastes pose as great a problem, if not a greater one, as municipal sanitary wastes if they are allowed to reach waterways in an untreated or uncontrolled state. The OWRC co-operates with industry in its efforts to find solutions to industrial waste treatment and disposal practices. The OWRC has regulatory powers which enable it to require industries to take specific actions on problems outlined by the Commission.

660. If such requirements are not met within a specified period of time, the OWRC can take the industry to court and, subject to adequate evidence being presented, can obtain a conviction under the *Ontario Water Resources Commission Act*. The assessment of fines and other damages is left to the discretion of the courts.

661. The OWRC was formed in 1957, shortly before ERCO had purchased the plant from the Dominion Fertilizer Company. Consequently, it has little information in its files regarding the activities of that company prior to 1957. Mr. Dennis Caplice, Director of Industrial Waste, gave testimony on behalf of OWRC and, as well, prepared for the Committee a report on the activities of his Division in the Port Maitland area from 1957 to 1967. The contact with ERCO dates back to May 10, 1957, when the OWRC was advised that ERCO intended to locate in Sherbrooke Township. On May 21, 1959, ERCO received a report from Canadian British Engineering Consultants concerning the regional water supply which would be available to ERCO.

662. From 1961 onwards, the OWRC expanded its Industrial Wastes Division so that regular surveillance of industries could be carried out. Their prime concern, as emphasized by Mr. Caplice in his testimony, was:

> "... in industrial water pollution control and we deal only with that respect of pollution that is waterborne away from the plant. We do not cover the other areas of pollution in these plants."

OWRC learned on August 1, 1961, that the Department of Lands and Forests had reported vegetation damage in the Port Maitland area, "The area affected corresponds roughly to that caused by drifting smoke from the ERCO plant." This report was tabled shortly after ERCO installed its wet phosphoric acid plant and it was at this stage that the OWRC became actively involved with ERCO's water pollution problem. On the basis of present-day legislation, a plant would have to deal with OWRC before plant start-up.

663. As a part of the standard operating procedure, the OWRC scheduled routine investigation in October, 1961, December, 1961, and January, 1962. Following these investigations, a report of ERCO's operations was prepared, including recommendations and conclusions. ERCO received the report. The major points were as follows:

(a) Samples were taken of waste water discharges from the plant to any receiving waters (creeks, rivers, lakes, etc.). A figure of 4.2 tons of fluoride per day (effluent from lagoons) was estimated to be discharged into Broad Creek. As mentioned in a letter dated February 5, 1968, this value of 4.2 tons per day was based on the analytical results of samples collected, and the maximum possible waste flow of 8.4 million gallons per day, which is the amount of water

that ERCO can take from the joint water system. It was later learned that the actual water consumption and waste flow at that time was 2.9 million gallons per day, and thus, the fluoride losses were estimated at 1.4 tons per day.

(b) It was known that there was a high fluoride content in the final plant discharge and the question of neutralizing such content was mentioned. A recommendation was made to treat hydrofluoric acid with lime, over a sufficient period of time, so that neutralization might be more complete. This was considered in a discussion between ERCO and OWRC. The problem was that the complex fluorides had first to be hydrolyzed to hydrofluoric acid, and then the acid could be neutralized with lime to produce the relatively insoluble calcium fluoride. ERCO's research group felt that the hydrolysis step required approximately 30 minutes contact with lime. If this contact time was not provided, the waste and lime mixture would reach the lagoons and the lime would settle and thus not be available for the reaction. Subsequent hydrolysis would produce hydrofluoric acid, and thus the pH of the effluent would drop, and the fluoride concentration would increase as the waste passed through the lagoon.

664. Subsequently, between February, 1964, and September, 1965, the inspections of the effluents from the ERCO plant were made by the OWRC. Fluoride levels varying from 15.0 to 170 p.p.m. were found in the final discharge from the lagoon into Broad Creek. Fluoride levels further downstream were around 1 p.p.m.—a very acceptable level—however, it was recommended by OWRC that a better neutralization system be devised by ERCO and that the lagoon dikes be raised so the wastes could be kept under water.

665. ERCO proposed a hydrofluoric acid recovery system. There was a market for this material, for fluoridation of municipal water supplies, and the recovery of this material would lessen the hydrolysis and neutralization problems being encountered in the waste treatment system.

666. On November 3, 1964, the OWRC again reviewed ERCO's waste treatment program and concluded that there was no significant change in the quality of the effluent. ERCO was, therefore, requested to submit to OWRC, within three months, a comprehensive treatment proposal. On April 7, 1965, officials of ERCO, at a meeting with OWRC, outlined a general program of waste treatment

that would involve a total expenditure of $590,000 before August of that year. On April 28, 1965, ERCO submitted detailed proposals for the reduction of water pollution at the Port Maitland works. They included a plan for recovering hydrofluorosilicic acid from their process, and a plan to transfer effluent from the fertilizer plant to the acid plant where the neutralization process could be improved. (The neutralization system already existed at the acid plant.) The proposal was to improve its operation and also pump the wastes from the fertilizer plant to it. They also proposed to install dust collectors inside the plant proper, to double the capacity of the lagoon areas, and to improve the neutralizing equipment. By September 14, 1965, there was still no improvement in the effluent from ERCO plant. At this stage, ERCO submitted a proposal for recirculation of effluent in the lagoon system, which might reduce to an acceptable limit the amount of fluoride reaching the Grand River. (OWRC had recommended for some time that ERCO effluent should not increase the river concentration of fluoride to more than 1 p.p.m.)

667. In March, 1966, OWRC again reviewed an industrial waste survey of the ERCO operation. It was felt that the effluent from the plant to Broad Creek was still not acceptable, despite the alleged efforts of ERCO to reduce the level of impurities. Figures from 33 to 682 p.p.m. of fluoride were cited as the range of samples taken at the point of entry into Broad Creek. About this variation, Mr. Caplice said:

> "I would like to point out here that the nature of this effluent, through the years that we have been sampling it, 1961 through 1966, was very variable in character. It appeared that you could catch it on a good day and you could catch it on a bad day. Much of it has to do with this business of how high the dikes are."

There are many factors that can influence the quality of effluent and consequently the interpretation of sampling data is not a simple procedure.

668. ERCO, by this time, had been piping effluent from the fertilizer plant to the acid plant and a new scrubbing system had been installed which was designed to reduce total emission of air pollutants from 3,000 pounds a day to 300 pounds a day. However, since most in-plant controls utilize "scrubbing" techniques—treating gaseous substances by water—any decrease in air emissions is countered by an increased amount of the pollutant in the water

effluent. This "new" effluent may contain pollutants that were once released to the atmosphere. Mr. Caplice stated:

> "The company feels that this increased water pollution load can be handled by existing treatment facilities."

Further investigations by OWRC in June, 1966, revealed that a reduction had actually occurred in the level of fluoride in the final effluent. However, the pH and phosphorous levels still were not satisfactory and suspended solids in the lagoon discharge were considered to be a problem. Later, on August 15, 1966, ERCO officials and OWRC staff met to review the effluent treatment program. Proposals for immediate and long-term control were discussed and ERCO agreed to present a formal plan covering these problems by December, 1966. An interim brief was submitted on September 13, outlining the conclusion of the previous meeting. During this interval, the Department of Lands and Forests reported concern over the level of lagoon water on ERCO property. This was investigated by OWRC staff, and ERCO took immediate steps to raise the water level, thereby achieving a "better settling" of solid matter.

669. On April 3, 1967, OWRC again met with ERCO officials to discuss ERCO's failure to submit treatment proposals as agreed to on August 15, 1966. ERCO explained that expansion of plant facilities had occupied the time of ERCO employees who would be carrying out the long-term program. A date of August 31, 1967, was agreed to by ERCO for the submission of a final report.

670. Under Section 50 of the *Ontario Water Resources Commission Act,* there is a provision called a "Requirement and Direction." With the approval of the Minister of Energy and Resources Management, the OWRC may request industries to submit reports on specified problems and require the industry to undertake specific programs to correct any such problems. Failure to comply with such a "Requirement and Direction" is punishable by fines of up to $200 a day for every day of default. OWRC "Requirement and Direction" prepared on April 4, 1967, meant that ERCO would have to submit a final waste control program to OWRC by August 31, 1967, or be subject to penalty under the Act. Such a report was filed on August 17, 1967, thereby fulfilling the formal OWRC request. On September 14, 1967, OWRC again met with ERCO officials to discuss the proposed waste control program. It was agreed that, if immediate modifications in the industrial process produced an effluent satisfactory to OWRC, no immediate action would have to be taken on installing a recirculatory system in the lagoons. This had the effect that on October 16, 1967, ERCO submitted propos-

als to raise their lagoon dikes and change the location of the final outlet for plant effluents. These steps were aimed at reducing "channelling" in the lagoon areas. (The direct progress of wastes from point of entry to lagoon to the final discharge point had been resulting in adequate settling of effluent.)

671. On December 5, 1967, OWRC formally approved ERCO's modifications to existing treatment facilities and, at that time, the company stated that a total recirculation system would be installed. Engineering work would be completed by May, 1968, and would effectively eliminate discharge of wastes into Broad Creek and the Grand River. An evaluation of Phase I (raising of dikes and relocating of discharge) would be submitted to the OWRC by the end of June, 1968. If Phase I did not produce satisfactory results, ERCO would consider installing Phase II, (recirculation system) and this could be completed by October, 1968. These plans were confirmed at a meeting on March 21, and in a letter from ERCO dated April 22. The Committee was advised that Phase I, which was delayed by technical problems, was completed and operating on March 15, 1968.

672. During the hearings, mention was made of OWRC's dealings with Sherbrooke Metallurgical Company, the other industry in the Port Maitland area. Continuing inspections by the OWRC staff revealed that, as stated in evidence by Mr. Caplice:

> "There is no serious water pollution problem resulting from the operations of Sherbrooke Metallurgical."

673. The efforts of the Industrial Wastes Division of OWRC have always been focused upon reducing the fluoride and phosphate levels that were being discharged into "public" water sources by ERCO. The low pH content (high acid) of water was also a concern. To overcome these problems, ERCO claimed to have spent a total of $826,000, in the years between 1961 and 1967, towards the direct control of water pollution. Planned capital expenditures for 1968 totalled $260,000, the bulk of which was for installing a recirculation system which would eliminate the discharge of wastes from the lagoon system to the watercourse.

674. The control of the water pollution problem at ERCO is technically a difficult one. The OWRC staff, appreciating such circumstances, felt that ERCO's "attitude was good; it was positive. They were concerned with the problem; they were meeting with us; they had technical people who I think our staff felt were knowledgeable and understood our problem and our viewpoint." The technical problems involved in correcting "pollution" problems in any industrial setting are not easily resolved. OWRC felt that

the progress of ERCO towards adequate abatement of water pollution had been a steadily progressive one, and it is ERCO's stated hope that by July, 1968, water pollution from the ERCO plant will cease to be a continuing problem. OWRC has been far more tolerant in its dealings with ERCO than perhaps was warranted. To ensure the reduction of water pollution caused by ERCO, the OWRC officials feel that the proposed recirculation system should be installed as quickly as possible. They also advise people in the Port Maitland area not to use cisterns as a source of drinking water.

675. The "water pollution" problem associated with ERCO is being resolved in co-operation with OWRC, but the "dust" blowing off the lagoons is, of course, "air pollution" and therefore is the responsibility of the Department of Health. This is an excellent example of overlapping jurisdictions on what is essentially a single environmental problem—pollution from a particular industry in a particular area.

676. For some time, residents of the Port Maitland area had been concerned about the possible contamination of their water supplies. Sampling of water from wells, cisterns, and rainwater barrels, between May 17 and August 28, 1962, revealed a range of fluoride values between 0.2 and 3.3 p.p.m. and phosphate ranges of between 0.8 and 80 p.p.m. The analyses were carried out by the OWRC at their laboratory (Toronto), but as Mr. Caplice stated in his testimony:

> "We rely on the person collecting the sample to adequately describe the source of the sample and this is what is recorded on our result sheets."

Some 37 water samples from area sources were tested. One sample, submitted by Mr. William Siddall, showed a reading of 37.8 p.p.m. —the highest of all the readings. But, unlike the other samples, no information was received by OWRC as to the exact source or location of the sample. This is the type of difficulty that occurs when questionable sampling procedures require meaningful analyses and important interpretation. If, for example, the sample was derived from rainwater from a roof, one set of interpretations must be made; if the sample was taken from a rain barrel, it is important to know whether it was taken in a dry spell or immediately after rain. As Mr. Caplice stated:

> "Our own view on outside sampling is that we put very little reliance on it, unless we know the exact source and nature of it. It is so difficult, of course, to ascertain as to how the samples were taken."

677. The responsibility for the purity of domestic water supplies, wells, or cisterns, if humans are drinking the water, lies with the local Medical Officer of Health. The OWRC will analyse individual drinking water samples forwarded to that agency, but this is a courtesy to the public and is not a legislative requirement.

678. ERCO, early in 1968, undertook a plan to raise the level of dikes in the lagoon area, and is planning to insure a constant level of water in the lagoons, which will eliminate "channelling" of water and the "drying out" of the gypsum beds, which should reduce the dust problem which has plagued the area. The Committee feels that this problem should be a continuing priority of the ERCO management.

CHAPTER IX

"Air of Death"— CBC Film

679. On Sunday, October 22, 1967, at 8:00 p.m., the film, "Air of Death" was presented as a CBC special and was shown on Channel 6. A portion of the film dealt with "the health issue at Dunnville" and, as mentioned in Chapter I, the Committee, together with its counsel and the counsel for the local "Air Pollution Committee" and for ERCO, viewed the film officially in the CBC studio on Thursday, February 22, 1967, at 10:30 a.m.

680. Much evidence was elicited during the hearings in respect of the film and its effects. It became very apparent that the impact of the film, not only locally, had been great indeed. Many people became convinced that their ailments were due to fluoride poisoning; vegetable products from the area were considered to be inedible and markets were lost; the name "Dunnville" became associated with dangerous pollution; persons shunned the area and economic loss was suffered. Fear, apprehension, and suspicion created a psychological effect which was very real.

681. The Committee was anxious to ensure that all evidence relative to the effects of fluoride and SO_2 in the area was available to it for study and assessment. It therefore requested the assistance of the CBC, which had said in its film, "For the past six months we've been researching this problem." On March 14th, 1968, the Committee wrote to Mr. L. Gosnell, the Producer of the film, attached a list of questions which were based upon statements made in the film, and asked him to supply answers to the questions. (Copy of letter is presented as Appendix IX.) This letter was acknowl-

edged by Mr. Gosnell on March 18, 1968, together with the statement that our letter of the 14th, "has been referred to the Corporation's executive for consideration." The Committee's counsel sent copies of the two letters mentioned here to Dr. George F. Davidson, the President of the CBC. Dr. Davidson's acknowledgement was dated March 22, 1968.

682. The original letter of March 14, addressed to Mr. Gosnell, was answered by Mr. Marce Munro, Acting General Manager, Network Broadcasting (English), in a letter dated March 22, 1968. Although a copy of this letter, as part of the complete correspondence is attached as Appendix X, it is pertinent to quote here portions of this letter:

> 683. "Quite apart from the terms of reference actually given to the Commission, we do not believe that a Commission established by a provincial jurisdiction could be given the authority to inquire into the preparation of a CBC program. As you know, the CBC has been established by Parliament for the purpose of operating a national broadcasting service and is accountable to Parliament for the conduct of its affairs and the discharge of its responsibilities."

> 684. In concluding his letter, Mr. Munro said:
>
> "Consequently, we do not consider that it would be in order for us to reply to the questions contained in your questionnaire."

685. This reply was a disappointment as we felt that the CBC, from its research studies on the problem, might have been able to supplement the information which we had on hand. A further attempt was made to solicit this help when the Chairman of the Committee wrote to Mr. Munro and to Dr. Davidson on March 27, 1968 (Appendix XI). Some excerpts from the letter to Mr. Munro will illustrate the Committee's concern:

> 686. "I wish to assure you that the Commissioners are most anxious to obtain all the assistance possible to understand and to assess this most complex and significant problem, and subsequently to make realistic recommendations which will help prevent and to control comparable situations."

> 687. "That portion of the film dealings with pollution in the so-called Dunnville area, contained results of your research which are of extremely great interest to the Commissioners, varying, as many of your results did, with some of

the evidence obtained at the Public Hearings and with the reported scientific literature."

688. "Citing a few examples of the divergence of your research results—which give the Commissioners great concern—may indicate the efforts which we are making in order to report fully and in an unbiased fashion the results and conclusions of all relevant research studies. I appreciate that you must have such evidence or the CBC would not have made the statements which it did."

689. In his letter, the Chairman then quoted a few statements made in the film, each of which was followed by a brief résumé of the evidence received by the Committee and which in each case differed from the CBC presentation.

The letter continued:

"These few examples, which cause us some perplexity, should suffice to indicate that unless we receive substantiated contrary evidence, our report will of necessity be contradictory to many of the statements made by the CBC.

"Naturally, therefore, it is most desirable that we solicit your assistance and benefit from the studies which your colleagues have made relevant to the problem in the Port Maitland area. It was for these reasons, and in a collaborative spirit, that the Committee asked Mr. Gosnell for his help through providing answers to the questions contained in the questionnaire submitted to him with the letter of March 14th. In this letter we asked if the answers to the questions could be received by April 10, 1968." (This letter was posted on March 27, in Toronto.)

690. The whole questionnaire, as mentioned, is to be found in Appendix IX. A few statements, which are quoted hereunder from the film, will indicate the positive position taken by the CBC in dealing with this very complicated problem. They will underline, too, the concern of the Committee which, it will be recalled, had many special consultants in all relevant fields of inquiry to assist it.

(a) "they (air pollution) cut a hole in your throat"
(b) "lung diseases as a whole are now the number-one killer in Canada"
(c) "from some of the richest farming land in the country"
(d) "dwarfed and shrivelled the grain"
(e) "damaged everything that grew"
(f) "grain yields cut in half"

(g) "fluorosis—swollen joints, falling teeth, pain, until the cattle lie down and die—hundreds of them"

(h) "about the health issue at Dunnville"

(i) "what's going to happen to these people who have fluoride poisoning"

(j) "... Russia has imposed the highest standards of purity in the world."

691. Let us now consider these statements in the light of evidence received by the Committee and of the scientific literature made available to it.

(a) "they (air pollution) cut a hole in your throat."

No clinical, surgical, pathological or postmortem examinations, as recorded in the world's relevant literature, have ever revealed such a condition which can be attributed to air pollution.

(b) "lung diseases, as a whole, are now the number-one killer in Canada."

The following table, reproduced from *Special Report Number 29, 1966,* published by the Medical Statistics Branch, Ontario Department of Health, lists the "Chief causes of death by sex, all ages, Ontario 1960-64." The Dominion Bureau of Statistics provides comparable complete vital statistics.

(c) "from some of the richest farm land in the country."

Evidence submitted by Professor Webber showed, "In parts of Haldimand County, soil productivity is impaired by inadequate soil drainage as a result of soil texture and structure." In Professor Clark's evidence, it is stated: "Most of the southwestern Ontario counties have both the favoured climate and soils which respond easily to intensive management. Therefore, a comparison of Haldimand County accomplishment with the average of the similar climatic areas in the province indicates that farmers in Haldimand have not yet achieved the average results." In the ten-year period, 1957-67, the yields of winter wheat, of oats, of fodder corn, and of hay in the Haldimand County at no time equalled the yields in the whole of southern Ontario, including Haldimand. In the one year 1963, the Haldimand County yield of shelled corn exceeded the yield for all of Southern Ontario.

TABLE XXVII

CHIEF CAUSES OF DEATH IN ONTARIO
1960-1964

Rank	*Causes of Death*	*Percent of Total Deaths* *Male*	*Female*	*Total*
1	Diseases of heart	40.4	36.5	38.8
2	Cancer	15.8	17.9	16.7
3	Vascular lesions of central nervous system	9.6	14.7	11.8
4	Accidental causes	7.7	4.6	6.4
5	Pneumonia, bronchitis & influenza	4.9	4.8	4.9
6	Diseases of the arteries	2.2	2.4	2.2
7	Congenital malformations	1.5	1.9	1.7
8	Immaturity	1.5	1.6	1.5
9	Diabetes Mellitus	1.1	1.7	1.4
10	Suicide & Self-inflicted injuries	1.4	0.5	1.0
11	Postnatal asphyxia & Atelectasis	1.0	0.8	0.9
12	Cirrhosis of liver	0.9	0.7	0.8
13	Birth injuries	0.8	0.8	0.7
14	Nephritis & Nephrosis	0.7	0.7	0.7
15	Ulcer of stomach & Duodenum	0.9	0.4	0.7
16	Hernia & Intestinal obstruction	0.6	0.7	0.7
17	Gastritis, Duodenitis, Enteritis (etc.)	0.4	0.5	0.5
18	Hypertension without mention (of heart)	0.4	0.5	0.5
19	Diseases of Prostate	0.6	—	0.4
20	Tuberculosis	0.4	0.2	0.3
	Total:	92.8	92.1	92.5
	All other causes	7.2	7.9	7.5

(d) "dwarfed and shrivelled the grain."

(e) "damaged everything that grew."

(f) "grain yields cut in half."

Considering (d), (e), and (f) together, the testimony given at the hearings, and which may be found in Chapter IV, speaks for itself. Admittedly, there has been some crop damage, but the above statements are obviously untrue. In particular, reference may be made to Mr. Paisley's statement about his corn. "I did good, it was the second highest

in the county," and about his oats, "Well, it was pretty good for the average of the county, I think it was 40 or 50 bushels to the acre." Mr. Rittenhouse said, "I haven't seen anything around our place, well, we have had no difference in any crops. They may be loaded with this here trash, whatever it is, but as far as growth is concerned, I have never seen anything wrong at all. No, as I said before, I see no difference in our crop." Mr. Harvey told the Committee, in evidence, "As far as corn goes, we haven't seen any bad results at all, our corn has been good as far as growth and yield." He also stated, "We had a field of barley and I never noticed anything in the cereal grains, our wheat was good this year." Mr. Niece, who grows wheat, oats, and barley, stated that his crops were "always good up to last summer, when we had one failure of oats [one field]." In this connection, Professor Tanner had stated, "Last year in much of Ontario or many parts of Ontario, the barley crop was one of the poorest barley crops we have had for some time and there was a fair amount of the diseased barley [yellow dwarf] also prevalent in Ontario." (See also Figure 7.)

(g) "fluorosis—swollen joints, falling teeth, pain, until the cattle lie down and die—hundreds of them."

The evidence has clearly demonstrated that no teeth of any cattle have "fallen" out. Three cattle were shot because of severe lameness, but cattle did not lie down and die, not even one, let alone hundreds of them.

(h) "about the health situation at Dunnville."

Not only is there no "health issue at Dunnville," but the total evidence has established, to our satisfaction, that there was no abnormal or unusual human health hazard in the "polluted" area of Port Maitland.

(i) "what's going to happen to these people who have fluoride poisoning."

There is no evidence that indicates that any "people have fluoride poisoning" in the Port Maitland area.

(j) "Russia has imposed the highest standards of purity in the world."

To quot from Dr. Martin's brief, as recorded in the evidence, "On the other hand, Lindberg (1964) reported atmospheric fluoride concentrations from 0.458 mg. per cubic

> meter near a superphosphate plant in the U.S.S.R. [Russia]. This would suggest maximum possible adult intakes of from 1.9 mg. to 9.7 mg. and high levels of fluoride intake were, in fact, confirmed by the finding of dental fluorosis and low incidence of dental caries in school children in that area." Standards and performance are not necessarily synonymous.

692. The fact that evidence, which was contrary to some of the statements made by the CBC in its film, was known by the CBC but not used, can be ascertained by reading the transcript of evidence. To cite specific instances would simply belabour the point. The Committee must say that it finds it difficult to understand why the CBC did not interview the local Medical Officer of Health, and other physicians who had attended the patients directly involved, and did not use any of the statements made to it by the one attending physician who was interviewed. The Committee also fails to understand why Dr. Marson, a consulting physician, who was the subject of comment in the film, was not contacted by the CBC until after the film was released. The inferences seem inescapable.

693. Evidence provided at the hearings by Mr. Camille Kneider, President of the Dunnville Chamber of Commerce, shows that the CBC disregarded pertinent statements by this official (Volume II, page 273):

> "The only thing is the distortion of news that was caused by the news media, especially CBC, who contacted me, came and contacted me and the Mayor of Dunnville—His Worship Charles Lundy—at the Victoria Hotel. They came specially, with a crew, and they asked about comments about the publicity, and we told them the way it was adverse publicity and we would like to rectify it, and they said that was what they came for, to rectify it. We decided on the questions and answers and we went outside the Victoria Hotel, right at the front of the Victoria Hotel, and he asked us a question. He asked me for my comments for about three or four or five minutes, and also the Mayor for a couple of minutes, and he told us that at 11:00 o'clock at night we would see it on the news. We did not see anything on the news that night, and then the following night and the following night, and my comments were not on at all. Then I was on for about half a second and somebody else was on for about five minutes."

It was known to the Chamber that monetary awards had been made to farmers located close to the Industries for alleged cattle and crop

damage. It was not until October of 1967, following the CBC "Air of Death" television presentation and subsequent newspaper releases, that rumors reached the Chamber that area residents were complaining that their businesses were being affected by unwarranted publicity and statements that the Dunnville area was polluted. The farmers directly involved, to whom compensation was paid, are at least five miles from Dunnville and this town was in no way affected by the alleged pollution. Furthermore, the supposed fluoride poisoning was not established judicially. We were therefore shocked to find the National Television, CBC, CFTO, and leading newspapers across Canada, issuing distorted, exaggerated news reports as if this town was directly involved. This untruthful publicity was not retracted. Although the Mayor and the President of the Chamber (of Dunnville) were personally interviewed, their statements were not published. On the contrary, the false publicity, insofar as this town is concerned, was repeated several times with emphasis. As a direct result of this publicity, residents of this town and area, not in the neighbourhood of the industry involved and not affected by any supposed pollution, have been seriously damaged. The Chamber has contacted some of these persons, but of course there must be many more of which we are not aware. (Mr. Kneider continued and cited the following examples.)

DICK BROS.

Dick Bros., R.R. No. 4, Dunnville, market growers, not affected by pollution, but unable to sell produce to markets or canners since "Air of Death" show. They normally have two market stands, one at Welland, and the other at Port Colborne, both about 24 miles east of Dunnville. Residents of these cities would not buy. Negotiations with canners to sell over 2,000 bushels of cabbage fell through.

MATHEW EDL

Mathew Edl, R.R. No. 1, Lowbanks, is a market gardener and has a stand in Dunnville and Niagara Falls (40 miles from Dunnville). On Saturday, following "Air of Death" show, he returned from the Niagara Falls market with 90 per cent of his produce. Residents of Niagara Falls said they could not buy from him anymore for fear of pollution. Says he does not know what to plant for 1968, and is afraid he will not be able to sell produce in 1968.

ELROY FARM MARKET

Fruit and Vegetable Store, Dunnville (lives on farm, R.R. Moulton). Had farm up for sale. Had agreement, with deposit, to sell farm prior to "Air of Death" show. Purchaser did not finalize sale.

MRS. G. CROSS

Pot Luck Acres, Fruit and Vegetable Highways Stand, R.R. No. 8, Dunnville. Closed two weeks after "Air of Death" show, no sales. People would not stop to buy any more. Stand at Port Colborne market—could not sell truckload of potatoes after "Air of Death" show. Has had to insulate garage to store potatoes over winter, does not know if they will sell in the spring.

MRS. STANLEY KOCJAN

R.R. No. 1, Dunnville. Had verbal agreement from Toronto buyer to purchase their farm week before "Air of Death" show. Following the "Air of Death" show, the buyer phoned and said he would not buy in polluted area.

MR. CLAIR KINDY

Reeve of Rainham Township and furrier by trade. Received a phone call from client in Welland area who wanted to know of another route to Selkirk to avoid driving through Dunnville. He sent coat by express.

MR. LLOYD McDANIEL

R.R. No. 9, Dunnville. Following "Air of Death" show, took 10 steers to Kitchener auction where normally he would sell all he takes. Had to return home with all 10 steers as no one would bid on them. Auctioneer had made mention that steers had come from Dunnville.

MRS. SHARON KLACKO

138 Cross Street East, Dunnville. Was waitress at Homeside Fish and Chip Restaurant. Following "Air of Death" show, a couple came in for a meal. The lady complained that the coffee was bad. The proprietress, Mrs. Stubbins, gave her a fresh cup of coffee. Lady complained it was just as bad. Mrs. Subbins was sorry, but there was nothing wrong with the coffee. The lady angrily chided her husband for stopping in this polluted town. These people were obviously tourists.

Mr. Kneider: *So that is the way things are in Dunnville—*
(p. 281) *that is the reason.*

694. The Committee has no other alternative but to record that unwarranted, untruthful, and irresponsible statements were made by the publicly-owned and publicly-financed Corporation, the CBC. They treated a complex problem in a way designed to create alarm and fear. Their treatment was not in keeping with the standards which the public is entitled to expect from the Corporation.

695. As a responsible Committee attempting to assess a difficult and complex problem, we are unable and unwilling to conclude our comments on the CBC production entitled "Air of Death" without one further reference to the "Dunnville" problem. The evidence before us makes it crystal clear that the "pollution" problem related only to the immediate area of Port Maitland and that no damage was caused to vegetation or livestock in the immediate vicinity of Dunnville. Through the careless use of the name "Dunnville" the residents of that area have suffered financial loss, which is just as real, and more easily identified, as the losses suffered by those within the "polluted" area. The residents of the "polluted" area have received compensation. Presumably the Dunnville residents will take such action, through the courts, as they may be advised.

CHAPTER X

The Role of the Ontario Federation of Agriculture in the Port Maitland Problem

696. The Ontario Federation of Agriculture has, for many years, played a major role in representing a large majority of the farmers in the province and in seeking solutions to their ever-increasing problems. Over the years, it has established a reputation as a responsible body whose officers have served the farming community well. In so doing, they have performed a great service to all aspects of life within the province.

697. In connection with the Port Maitland "problem," the OFA carried out its traditional function, as one would expect. Not only did it provide effective support to the farmers involved, in their efforts to obtain compensation for their damages and losses, but it was also instrumental in having this Committee of Inquiry established.

698. In the light of the above, it is with regret that the Committee is, in the interest of accuracy and on behalf of the residents of the Port Maitland area, compelled to take issue with certain activities of and statements made by or attributed to certain officers of the OFA. It would be manifestly unfair if the statements made by Mr. D. W. Middleton, in giving evidence before the Committee and in correspondence or press releases, were allowed to go unchallenged. In all of these, he purported to speak on behalf of the OFA. The Committee is also concerned about the accuracy

of a news release to *Farm News,* attributed to Mr. Munro, the President of the OFA. Our areas of concern are set out hereunder.

699. There is little doubt that Mr. Middleton was, to some extent, involved in the production of the CBC film, "Air of Death." His motives remain unclear, particularly in view of the inaccuracies in the film, as demonstrated in Chapter IX. The following statements are quoted from his testimony to the Committee:

> "We in fact screened the film and had a showing of it before it went on the air. We were aware of what was going on and I don't in any sense hide the fact that we were involved in this insofar as our organization could be."

And again:

> "For my purposes—let us not kid ourselves—the film was well designed to get public awareness of a pollution problem, well designed to accomplish its mission."

700. As a result of questioning by one of the Commissioners during the hearing, Mr. Middleton felt impelled to write to the Commissioner on March 22, 1968, from which we quote as follows:

> " . . . I am sure you recognize there would have been no commission or inquiry without the publicity created by the CBC broadcast . . ."

Copies of this letter were sent to the Ministers of Health and of Agriculture and Food, and to the President of the OFA. A memorandum summarizing its contents was mailed to the presidents, secretaries, and members of the OFA, and, as well, an item headed "For Immediate Release" was sent to the *Farm News.*

701. The facts, as they have been made evident to the Committee, are that Mr. Middleton attended a meeting in the office of the Minister of Health on August 22, 1967, called to discuss the problem of pollution in the Port Maitland area. As a result of the representations made on behalf of the OFA, the Minister undertook to set up an objective Committee to study the matter and, two days later, on August 24, 1967, Cabinet approval was given to the appointment of Dr. G. E. Hall, as Chairman of the Committee. Mr. Middleton also stated in evidence that his organization had been asking for a pollution conference in the Province which was not forthcoming, whereas in fact the Prime Minister of Ontario had, on July 25, 1967, publicly announced that a general conference on pollution would be held in Toronto on December 4, 5, and 6, 1967.

702. While recognizing the fact that the OFA was instrumental in obtaining compensation for the farmers involved, and in the establishment of this Committee, the Committee can find no justification for Mr. Middleton's involvement in or approval of "Air of Death." His actions and statements, however motivated, were not in keeping with the traditions or best interests of the OFA.

703. In the course of his testimony before the Committee, Mr. Middleton made the following statement, which was challenged by one of the Commissioners and which subsequently led Mr. Middleton to write the letter referred to in paragraph 700. The statement follows:

> "It was the decision of the OFA Executive and, in fact, in agreement with the company and with the Department of Health, that there would be no publicity on the problem we had at Dunnville."

704. In his letter to the Commissioner, referred to in paragraph 700, the second paragraph reads as follows:

> "Your point of shock is a valid point, but I think you must realize that there were, in fact, three parties to the 'no publicity' agreement and that all three share equally in any responsibility there is for what you intimated was an undesirable silence."

705. The other parties to the alleged agreement, namely the Department of Health and ERCO, deny that any such agreement existed. The Committee rejects the evidence of Mr. Middleton. Not only is there a preponderance of evidence against the existence of any such agreement, but the Committee finds that Mr. Middleton knew, or should have known, that science had long since established that milk from dairy herds pasturing on fields polluted with fluoride was perfectly normal. The Committee cannot understand why Mr. Middleton found it necessary or desirable to express the view that such a "secrecy agreement" existed.

706. It is especially regrettable that Mr. Munro, the President of the OFA, is quoted in the OFA release to *Farm News* as saying:

> "Mr. Middleton said—and I heard him—that we wanted to prevent a scare on the part of the uninformed."

and:

> "The Department of Health had done research in the area and said there was no danger."

707. Not only have representatives of the other parties to the alleged agreement denied the existence of such an agreement, but their denials can be fully supported by the fact that the two quotations above contradict each other, and by the further fact that Mr. Middleton knew, or should have known, as an officer of the OFA, that science had long since disproved the view that milk of fluoritic animals was high in fluorides. In actual fact, the transcript of evidence does not contain any such statements as Mr. Munro attributed to Mr. Middleton.

708. The Committee regretfully concludes that the best interests of the OFA were not well served by the statements made by Mr. Middleton before, during, or after the public hearings of the Committee.

709. As stated at the beginning of this chapter, the OFA is an effective and responsible organization which has done its job well but, in our opinion, it deserves better and more responsible representation.

CHAPTER XI

General Discussion

710. The problem of air, soil, and water pollution is of great complexity and has significant and far-reaching socio-economic implications. It is primarily a man-made problem. Its solution is one which requires knowledge, understanding, compromise, and planning in varying degrees; pollution is a companion of industrialization. Its prevention or control cannot be studied without adequate scientific personnel; its effects cannot be adequately assessed, and remedial programs initiated, without a knowledgeable, co-ordinated organization with proper laboratory facilities, and the whole backed up by concerned and dedicated research personnel in teaching and research institutions. In the Port Maitland area, some of these elements were missing. Confusion and inadequate communication engendered apprehension and concern in the minds of many persons.

711. In general, here is what has happened: Field members of the Department of Agriculture and Food first reported "damage" to crops in the area in 1962. The Veterinary Services Branch became involved through the Department of Health, and continued in the succeeding years. In July, 1965, personnel of the Ontario Veterinary College, of the University of Guelph, were consulted and confirmed the diagnosis of fluorosis in cattle in the Port Maitland area. It was not until January, 1968, that these same consultants, at the request of this Committee, became involved again in the "fluorosis problem" in the Port Maitland area.

712. The OWRC has been involved with ERCO from 1959, and the Environmental Health Branch of the Department of Health has likewise been involved with ERCO, on in-plant studies and

casual outdoor studies, since 1959. The Environmental Health Branch had fluoride determinations of vegetation and urine made from 1961 through 1968. Some samples of urine from cattle were taken by local private-practising veterinarians and sent to the Department of Health laboratories. Samples of human blood and urine were taken by several local consulting and Department medical personnel and sent to the Environmental Health Laboratory for analysis. Samples of livestock urine, etc., were sent to the Guelph Laboratories. Water samples were tested by the OWRC on various occasions over several years. In only a few instances, as revealed by the evidence, but not necessarily in fact, were the persons who took the samples, other than government officers, advised of the results of such fluoride analysis. Therefore, in turn, the results could not be discussed with the owner of the livestock or crops, or with the persons from whom blood and/or urine samples had been taken; one notable exception to this is reported in paragraph 259. (J. Casina was the only area resident tested by a government physician. He was notified by letter of the results in less than two weeks.) Some samples were taken by private physicians, analysed at Government Laboratories and the report sent back to the physicians. Naturally, such lack of knowledge of the results, and the absence of discussions with the persons involved, tended to create apprehension and ill-will. There was a lamentable lack of communication.

713. The strict adherence to the doctor-patient concept by the health agencies was a major influencing factor in the whole situation. It is sincerely suggested that the normal doctor-patient relationship, which is most desirable and indeed essential when there are only the two parties involved, is entirely unsatisfactory where there is a general health matter in question in the community. To rely on the individual doctor to communicate or not to communicate results, or to give assurances based on the results, is quite inadequate.

714. In essence, there was no totally planned approach to the pollution problem in the Port Maitland area.

715. In spite of the efforts of concerned officials, ERCO has been unwilling or unable to control effectively fluoride emissions from their plants. Since there would have been no fluoride problem in the area if such emissions had been controlled, a brief discussion of their efforts is in order.

716. The Electric Reduction Company filed a brief on January 26th, 1968, which outlined their total capital expenditure on

air and water pollution control equipment. Through Mr. Chester Duncan, P. Eng., Waste Control Engineer, ERCO also filed a brief on March 21, 1968, which gave a detailed breakdown of their present pollution abatement equipment as well as future plans for control equipment and procedures. The points are here summarized:

(a) Mr. Duncan felt that the Resources Research Report, (paragraph 34) was generally accurate, but he questioned the accuracy of measuring fluorides which are emitted from the curing pile building. The Air Pollution Control Service has reviewed the problem and feels that techniques for measuring curing pile emissions are accurate and can be used as a basic guideline.

(b) ERCO stated their intention to install a cyclonic scrubber on the calcium phosphate plant, which would reduce fluoride emissions from 70-80 pounds a day to 15 pounds a day. The Air Pollution Control Service feels that this is a reasonable and accurate figure in the light of available technology.

(c) Both single and triple superphosphate leave the curing "den" and are conveyed to storage piles, where the reaction continues for several weeks. During this "curing" period, fluorides (SiF_4) are continually emittted to the atmosphere and, to date, ERCO admitted that it has not been able to control such emissions. They have investigated the possibility of introducing a chemical additive which retards the "curing reaction" and allows the fluoride to evolve more slowly. Mr. Huffstutler, Florida State Board of Health, stated that some Florida industries seal their storage building and "place them under vacuum for scrubbing the off-gasses." (Huffstutler, page 1159.) Both techniques offer some measure of abatement for fluorides evolving from storage areas.

(d) ERCO has stated publicly that they will not manufacture any triple or single superphosphates during the growing season of May through September, which will temporarily solve this particularly difficult problem. However, it is only a temporary solution and, if market demand for these products necessitates year-round production, then the problem will again present itself. The Air Pollution Control Service feels that almost fifty per cent of total fluoride emissions come from the "curing" sheds and recommends

that some permanent technique be instituted to meet this problem.

(e) The "den" area, where acids are mixed with phosphate rock, has two scrubbers in place, which ERCO feels are not adequate. Mr. Duncan stated that the company intends to install a third scrubber, if and when market demand increases and the company resumes superphosphate production. Again, this is a temporary step in terms of solution. The design work for the third scrubber is complete, and the Committee fells that all attempts should be made to install controls or modify processes before a problem begins. Surely the difficulties ERCO has had to date would lead management towards permanent planned abatement programs.

(f) The gypsum ponds or lagoons present a dual problem. The release of too much effluent into water courses results in water pollution. An insufficient amount of water in the lagoons causes a "drying out" and formation of dust, which then becomes an air pollution problem. ERCO has raised the lagoon dikes and has stated that they will install a pond "recycling" system which would (i) eliminate any water effluent from leaving ERCO property and (ii) ensure a sufficiently high water level to prevent dust "blow-off." This system is scheduled for installation in late 1968, or early 1969. The Air Pollution Control Service feels that if a sufficient water level is maintained in the lagoons, then this possible source of air pollution will not constitute a serious problem. The OWRC feels that the immediate installation of a recycling system will effectively eliminate water pollution.

(g) It should be noted, in connection with the "lagoon" problem, that problems with dust have occurred mainly during the winter. Mr. Duncan stated that, if the suspension in the lagoons were not covered by water, the material would freeze, break into a fine powder, and would be blown around the countryside. He stated that "If it [lagoon] is completely covered with water and frozen . . . if there is ice on top of it, there is not a problem—only if it [lagoon] is not covered by water." By installing a recycling system and by maintaining a sufficient water level, ERCO should be able to control both water and air pollution problems from this source.

(h) Mr. Huffstutler, in his testimony, described in detail the system of allowable emissions of fluoride that the Florida State Board of Health has instituted. At one time, the State allowed 0.6 pounds of fluoride per ton of P_2O_5 produced, if the plant has a "producing" operation. New plants would be limited to 0.4 pounds of fluoride per ton of P_2O_5. Other existing plants must maintain a maximum figure of 0.4 pounds per ton of P_2O_5; the Air Pollution Control Service suggested that ERCO had the figure of 200 pounds a day in terms of allowable fluoride emission (July 28, 1967). Mr. Duncan agreed that such a figure of 200 pounds per day was an "achievable" figure. "All our problems exist in the pile. I think if we control the pile, we should meet fairly close to these limits." (Duncan, page 1630.) Both Mr. Drowley, Chief, Air Pollution Control Service, and Mr. Duncan agreed that the "curing piles" do constitute the greatest potential pollution hazard, and the Committee feels some definite procedures should be instituted whereby the fluoride emissions from the piles are reduced.

717. Although some damage to vegetation in the Port Maitland area was evident as early as 1962, no complaints of damage to farm animals were made until the summer of 1965. The "limed" candle readings made in the area showed a dramatic increase in fluoride concentrations and, at some stations, ten-fold increases were noted. The Committee concludes that there was a definite relationship between the sharp increase in fluoride emissions from ERCO and the increase in vegetation damage and "crippling" effects on some farm animals after 1965.

718. As a result of this damage to vegetation and farm animals in 1965, the local farmers and ERCO agreed to arrangements for a system of compensation for agricultural damage, to be made by an independent assessor. A summary of these activities follows:

(a) Assessments of damage to livestock, crops, and other vegetation, for the crop year 1965, were made in the spring and the early summer of 1966. There are many difficulties in attempting to assess crop damage when there has been no opportunity for the assessor to see the crops in various stages of growth, no opportunity to observe the soil and crop management practices, e.g. varieties used, cultivation, seeding, fertilizing, weed control, etc., and few opportunities of examining well-kept records. Nevertheless, the assessments were made in good faith and with compassion.

The terms of reference were certainly general, leaving a great deal to the judgement and discretion of the assessor.

(b) When any animal was considered by the assessor to be affected by fluorosis, not only was compensation for the animal paid, but the owner was permitted to retain the animal. If the owner decided to dispose of the animal, the sale price of the animal was his; if he decided to retain the animal in his herd, that was his decision and gain. In fact, there are some cows, compensated for in 1965, still in herds in the area, having had subsequent calves and still producing milk in 1968.

(c) Assessments of damage for the crop year of 1966 were made during 1967, under the same general conditions. The Committee considers that the awards made were generous and, in some instances, more than generous. One such example should be mentioned. In 1966 (for 1965), Mr. ————, with a herd of 50 cows, was awarded full compensation for his entire herd of cows, which in the opinion of the assessor was justified. He sold 10 of these cows and replaced them. The other cows were retained in his herd. In 1967 (for 1966), this same farmer claimed for 10 cows, these being replacements which he had purchased in the preceding twelve months. The awards were granted. Thus, in a period of two years, this farmer had been compensated for his entire cow herd, including replacements. Yet only individual cows in his herd showed symptoms of fluoride toxicosis. His evidence showed that forty per cent of his present cow herd were cows on which he had received compensation for the crop year 1965, and which were still producing and reproducing normally in 1968.

(d) A perusal of the records of claims and of assessments for the crop year 1965, and for the crop year 1966, give convincing evidence of the leniency of the assessors and the willingness of ERCO to compensate for almost any alleged damage and loss. At the same time, the impression has been left with the Committee that not a few claims were presented and awards made which were quite unjustifiable.

(e) It should be pointed out at this time that ERCO accepted a further degree of responsibility in connection with the possible contamination of hay crops in the "isopleth area." Mr. Pepper, counsel for ERCO, stated at the hearings

(Volume IV, p. 530, line 14), "I was concerned about the cattle; anybody would be. We don't think there is any occasion for any damage to cattle during this winter, but we have been a bit bothered that the farmers might be using hay from previous years when our anti-pollution control devices were not as good as they are today and, therefore, the company has obtained hay from outside the Dunnville area and is presently providing hay to such farmers as have cattle and who are disposed to take it, and that is something we are doing at the moment."

(f) ERCO also accepted responsibility for claims to glass in the area. Mr. Pepper stated at the hearings, (Volume IV, p. 529), "Within a short radius of the plant, ERCO accepts a responsibility and instructs me that it intends, in the spring of this year [1968], to replace the glass of anybody who can persuade us that they have been seriously affected."

(g) The mere fact that claims from area residents were solicited by ERCO, and that awards were made on the assessments of such claims, established the premise that "damage and loss" has been experienced in the area as a result of the alleged pollution. Evidence to support such a contention was presented, not only by some of the area residents (even though the degree of alleged damage is questioned in some instances), but by evidence presented by the Department of Health and by the Department of Agriculture and Food.

719. Since ERCO has not been engaged in the manufacture of single or triple superphosphate since the beginning of the crop year 1968, the likelihood of continued agricultural damage and/or loss is reduced, since it is this source (superphosphate plant) which accounts for approximately 50 per cent of all emissions of fluoride to the atmosphere. ERCO has decided not to continue the present system of arbitration for the crop year 1968, but has promised to make on-the-spot assessment of any complaint by an area farmer. This procedure, we feel, will be more effective, since the damage can be evaluated on the basis of existing conditions, something that was not possible when assessors in previous years had to evaluate damage in the winter of one year for the period of May through September of the previous year. The experiences in the area, for the year 1968, should be reviewed by the several groups concerned.

720. The evidence which was presented, showing that dust and fumes from the plant(s) were a nuisance and had deleterious

effects on various materials and objects, was almost dramatic. The meteorological data, which the Committee had assembled for it, was necessary as corroborative information. The massive plumes of "smoke" from the stack of the ERCO plant left no doubt in anyone's mind as to the direction of the winds and the area which would be contaminated if pollutants were present in the "smoke." Likewise, to have seen a "cloud of dust" blowing off the lagoon in mid-winter permitted the Committee to follow the vivid evidence, as related to window glass, electric light bulbs, paint, and other objects and materials, with added knowledge. That ERCO, as previously mentioned, had agreed to compensate for damage to windows and other glass objects is heartening and a move in the right direction, but it does not replace adequate controls.

721. The whole question of permissible fluoride emissions—criteria, standards, objectives—should be a subject of separate examination. The Committee urges that immediate consideration be given to the question of adequate standards for detection and measurement of all pollutants, since it is only by the development of adequate enforceable standards that future problems like the Port Maitland situation may be avoided.

722. Any standard must be formulated with a clear view to the objective to be achieved. If the basic assumption is that emissions can never be entirely eliminated, the question then arises as to the levels of emissions which should be permitted. This, in turn, requires policy decisions on other questions, e.g. should the regulations be designed to prevent economic damage or would they be designed to prevent injury of all kinds, whether resulting in economic loss or not; should residents in the area of the fluoride-emitting industry be expected to adjust their agricultural methods and practices to a level of atmospheric fluorides, and if so, to what level?

723. The standard must be designed to fit a specific situation. The Florida regulations, for example, are expressed as maximum permissible emission rates, but the reason for such a standard is that the unusual concentrations of industrial sources of fluorides within a relatively small area would make it impossible to enforce any other kind of standard, since an offender could never be identified (Huffstutler, Volume VIII, p. 1174, lines 3-25). Again, even when the kind of standard has been selected, the possibility of meeting the standard, and therefore, the severity of the standard, must be fixed on the basis of local conditions. Mr. Huffstutler listed a number of factors to be considered in setting a standard, including

such things as climatic conditions, the nature of the industrial operation and its products, and activity in the surrounding area (Volume VIII, p. 1175, line 2; p. 1176, line 30). The point was made clear by Mr. Huffstutler that standards should not be exported unless it is certain that they fit the foreign situation.

724. It is submitted that, if standards are to be set, they should only be set after full consideration has been given to the factors enumerated by Mr. Huffstutler and others, and to the several questions posed in this report. A great deal of further work must be done, if reasonable and realistic standards are to be set for Ontario.

725. If standards of ambient air concentrations are to be set, it is necessarily implied that both industry and the enforcement agencies are capable of measuring the concentrations of fluorides in the ambient air at any time.

726. A standard must be directed to those fluoride compounds which are of significance with respect to injury to man, crops, livestock, etc. Mr. Huffstutler's evidence was that Florida regulations deal with gaseous or water-soluble fluorides (Volume VIII, p. 1173, lines 6-17). It follows that, unless equipment is available which is capable of differentiating and measuring gaseous or water-soluble fluoride compounds and water-insoluble compounds, a regulation based on the concentrations of such compounds in the ambient air is meaningless. It is clear from Mr. Drowley's evidence that neither the "limed" candles nor the automatic analysers are capable of such differentiation (Volume X, p. 1484, lines 3-6; p. 1486, lines 13-20). "There is little point in fixing a speed limit if neither the constable nor the citizen has a reliable speedometer." The Committee is compelled to agree with Counsel for ERCO on this point.

727. The question has been raised on several occasions as to why the Department of Health, or the Township of Sherbrooke, did not close the ERCO plant and insist on acceptable standards of emission being guaranteed before the plant could reopen. It is a complicated matter. Mr. William Siddall, the former Reeve of Sherbrooke Township, in his evidence gave part of the answer:

Mr. Gordon: *(Vol X, p. 1538)*	*What is your present occupation?*
Mr. Siddall:	*My present occupation is commercial fishing.*
Mr. Gordon:	*Do you hold any municipal office in this area?*

Mr. Siddall:	*I held the Reeveship for fifteen years until this year.*
Mr. Gordon:	*What is it you wanted to tell the Commission?*
Mr. Siddall:	*Well, I have heard a lot of evidence given here today from different fellows and doctors and everything else about the condition of things here. One thing, as Mr. Drowley said, on our by-law of the township that was passed in 1963, that was true, but we couldn't put that into force until 1965. Our hands were tied on that by-law. When 1965 came, I was going to try to enforce it, but I didn't think it was right for me to use the company's money and go after that company out of their taxes, of which they pay 75 per cent of the taxes in the township. I didn't want to use their money.*
Mr. Gordon:	*You mean the ERCO plant's money?*
Mr. Siddall:	*That is right, in taxes. They pay it through the township. Another thing, I didn't think we should take action and throw 350 men out of work who worked there. So I went to the plants and begged with them.*

728. Although Sherbrooke Township passed a by-law in 1963, which would allow the Township to control industrial emissions, the by-law did not come into effect until 1965; therefore, for a period of at least two years, there was no provincial or municipal legislation which would permit either the Township of Sherbrooke or the Department of Health to come to grips with the problem in the Port Maitland area. The Department of Health could not exercise real authority until 1967. The establishment of this Committee of Inquiry was in keeping with the new responsibility.

729. Mr. Siddall pointed out his personal dilemma when he mentioned that ERCO, in fact, was paying 75 per cent of the township tax bill. Certainly, it was responsible to be cautious in terms of instituting litigation against his largest single taxpayer in that township. This is an economic reality, which is appreciated by the Committe. Mr. Siddall was an honest man to place this question in perspective voluntarily. Mr. Siddall also related his many attempts to persuade ERCO officials to come to grips with the problem and he

relied, perhaps too heavily, on the willingness of the company to deal with the problem. Evidence has indicated that several of the technical problems were difficult to solve, but ERCO did not move quickly enough, even within the limits of current technological knowledge. The same general criticism can be voiced against the Sherbrooke Metallurgical Company.

730. Mr. Siddall stated that he felt it was the responsibility of the Department of Health to deal with such a large problem. In the past five years, the Department of Health assisted various municipalities in Ontario in drafting municipal by-laws which would allow for greater control of local industries. The history of municipal enforcement of by-laws, relative to pollution control, has not been a particularly successful one; ultimately the Department of Health was prompted, in 1967, to assume complete control for air pollution abatement activities in the province.

731. The answer to the question raised in paragraph 727 is that, until 1965, there was no enforceable by-law in the Township of Sherbrooke to control pollution and, after 1965, the municipal authorities were reluctant to close down their largest taxpayer. The Department of Health of Ontario had no authority to exercise in this connection, until 1967, when this Committee of Inquiry was established.

732. There are many questions which should be considered by the residents of the area, on an individual basis, relative to their farming operations. It is sufficient at the moment to say that farming continues to be profitable in many areas in the U.K. and the U.S.A., where comparable types of plants, with comparable levels of emissions, have been in operation for years. Certainly, the factories in question have a responsibility to meet reasonable emissions and pollution standards, and this must be insisted upon in the interest of the people, their comfort, and their livelihood.

733. Just as there are types of animals and varieties of vegetables and crops which are susceptible to fluorides, there are also types of animals and varieties of vegetables and crops which are much more resistant to damage at the same levels of fluoride concentration. Furthermore, from various chapters in this report wherein the present accumulated knowledge of this "problem" has been presented, it should be fairly apparent that some changes in farm practices and in farm management will produce continued prosperity in the area. The companies, of course, must play their responsible role in this readjustment which should, and indeed can, only be made with knowledge, with goodwill, with assistance

from the several agricultural and health agencies, and through adequate and continued communication.

734. The subject of the effects of the pollutants on human health has been presented and discussed in great detail. But here, once more, we wish to emphasize our concern for the several people of the area who felt that they were suffering from, or being affected by, fluorides in the air which they breathed, in the food which they ate, and/or in the water which they drank. We understand their apprehension, but it is still difficult, after these several months, to appreciate the attitude of one of the residents who, after being advised repeatedly by competent and recognized medical authorities that he did not have fluorosis, acted as though he was disappointed with the verdict. However, it was with great interest that we heard indisputable evidence which proved conclusively that none of the persons in the area, who had taken advantage of the offer to be hospitalized and examined by distinguished specialists, had any symptoms or signs suggestive of fluorosis. The people of the Port Maitland area can be assured that there is no human health hazard associated with pollutants being emitted from the industrial plants in the area.

CHAPTER XII

Summary

735. This report has dealt with many facets of a many-sided problem. The summary could, therefore, if treated in the conventional manner of recapitulating evidence and conclusions already dealt with in much detail, become another long chapter. We feel this is unnecessary since we assume that the body of the report will be read. Consequently, the summary is presented in the form of brief statements, the background and evidence for which are to be found in the relevant chapters.

736. The two major industrial pollutants in the Port Maitland area are fluoride and sulphur dioxide. Neither of these is present in the air in sufficient amounts to produce any danger to human health.

737. The fluoride concentration of domestic drinking water in the area is within normal limits and causes no danger to human health.

738. The consumption of meat, milk and vegetables, and other foodstuff produced or grown in the area, presents no danger to human health.

739. The amount of fluoride in the soil of the area is well within the normal range for agricultural soils; the soil is neither "polluted" nor has there been any decrease in soil productivity as a result of the addition of fluorides to the soil from the air.

740. High concentrations of fluoride in the soil do not lead to high concentrations of fluoride in plants grown on such soil.

741. By maintaining a minimum soil acidity level (pH 6), sulphur dioxide is rendered relatively harmless; it cannot accumulate in the soil as such, and therefore sulphur dioxide in the soil will have no influence on plants grown on such soil.

742. Damage to various types of vegetation on certain farms in the area has been caused by the air-borne pollutant fluoride. It is suspected that some damage has been produced by sulphur dioxide. Since the heaviest emissions of fluoride occurred in 1965, it may be assumed that, although some damage was reported as early as 1962, the damage probably reached a maximum in 1965. Although some fields of corn and cereal grains on some farms were below county averages, there were at the same time quite satisfactory and even excellent crops grown by other farmers in the same area.

743. In general, market gardeners in the area experienced considerable damage to many of their crops and suffered substantial loss as a result of air pollution.

744. Certain shrubs, trees, and flowers may have been affected by air-borne pollutants.

745. Some dairy cattle on some of the farms have developed fluorosis of varying degrees—this undoubtedly from consuming fodder contaminated with fluoride. In advanced cases, milk production has decreased.

746. Compensation has been paid by ERCO for alleged and actual damage to general vegetation, vegetable crops, field crops, cattle, and for decreases in milk production.

747. Although compensation has been paid for pigs in the area, no evidence was presented to indicate that fluorosis was a problem in swine.

748. It is probable that some sheep were affected by fluoride, although no positive diagnosis of fluorosis was made.

749. There is no evidence that poultry have been affected by fluorides. They are highly resistant to fluorides, yet compensation was paid.

750. It is highly improbable that sulphur dioxide has had any effect, directly or indirectly, upon area livestock.

751. There has been physical damage, in the area close to the plants, to paint, glass, metal fencing, chrome fittings, and other objects.

752. There has been a dust and odour nuisance problem from the plants and lagoons—the dust problem being sporadic but none the less accumulative.

753. While some steps were taken by ERCO to reduce the fluoride emissions from the plant and the lagoons, they have not done as much as they should have done or as much as is technologically feasible. In general, they did too little, too late. The cut-back in production, during the summer of 1968, is an expediency and a temporary alleviation; it is not a solution to the problem.

754. The Sherbrooke Metallurgical Company seemed indifferent to the pollution problem in the area, even though sulphur dioxide and zinc sulphide were being emitted into the air from their plant.

755. Various measuring devices have been used in the area to determine the concentration of pollutants in the air. The units of measurement are different for the different devices, making it practically impossible to compare the results of one method of measurement with those of another.

756. The difference in the effects of soluble and insoluble fluorides is very marked, yet no measurements of soluble versus insoluble fluoride have been made. This is obviously important in terms of damage to man, to animals, and to plants.

757. Damage to livestock, crops, etc., has been assessed through a procedure that could not possibly be reasonably accurate or just, e.g. (a) crop and vegetation damage was assessed the year after the crop was harvested and (b) animals were condemned on a herd or premises basis without the diagnosis of fluorosis being made on an individual animal basis. There are some cattle, in several herds compensated for by ERCO, which have freshened and are still good milk producers.

758. Some farmers in the area practising good farm management have produced crops equal to and, in one instance at least, better than the Ontario average.

759. ERCO, in 1968, instituted a program of hay replacement for area farmers, wherever the fluoride content of the farmer's hay was found to be high. Later, March 1968, ERCO assumed the responsibility for the replacement of glass windows, etc., which have been damaged by fumes and/or dust from their plant.

760. No farmer in Dunn Township indicated that any damage to livestock, to vegetable crops, to field crops or to general vegeta-

tion, as a result of air, soil, or water pollution, had been experienced.

761. Several market gardeners outside of the Port Maitland area and completely beyond any pollution, suffered real economic loss when, as a result of statements made by the CBC, they were unable to sell their products.

762. The area of so-called pollution was readily defined through the use of a polar plot and isopleths. It extended in a northeasterly direction, cone-shaped, from Port Maitland. Dunnville is about four miles northwest of Port Maitland and, of course, was far outside the affected area.

CHAPTER XIII

Recommendations

763. Since damage to animals, plants, and various materials has been caused by fluorides in the emissions from the ERCO plant at Port Maitland, it is essential that ERCO should install the necessary equipment and modify their operations to reduce dust emissions from the lagoons, and emissions from the curing sheds, to acceptable limits under full plant operation. The payment of even generous compensation is not a substitute for good operational control; compensation payments should be an emergency policy only.

764. Because of the potential health hazards to in-plant workers, ERCO should have a consulting physician associated with it to ensure the good health of its employees.

765. The Sherbrooke Metallurgical Company should become acutely aware of their pollution problem and should study the advisability of increasing the height of their stacks by a factor of 2.

766. It should be the responsibility of industry to measure and record the emissions of pollutants, etc., from their plants. They should be encouraged, for their own sake, to do so; if necessary they should be obliged to do so.

767. Meaningful standards should be established by government for the permissive concentrations of various pollutants in the air, relative to the effects on human health, livestock, and plants. The standards should acknowledge differences between rural, semi-urban, and urban areas.

768. Since the concentration of total fluorides (particulate, gaseous, insoluble, soluble) in the air does not reflect the potential hazard of the fluoride, it is essential that measuring devices be used, which will indicate the proportion of soluble and insoluble fluorides.

769. Special consideration should be given to the standardization of units of concentration used in reporting the quantities of pollutants in air, fluids, vegetation, and other substances.

770. It is essential, above all else, to assure that adequate personal communication exists between the respective government agencies, and between the government agencies and the individual people involved in an area pollution problem. Reasons for making tests, the results of the tests, and the meaning and significance of the results should be known by the persons concerned. Understanding and co-operation are absolute keystones in the resolution of the psychologically highly charged situation which engulfs a pollution problem.

771. The roles of the County Agricultural Representative and the Medical Officer of Health should be thoroughly re-examined, in the light of issues which arose in the current pollution problem in the Port Maitland area, and their terms of reference changed if necessary.

772. Because of the high pathogenic bacteria count reported from several cisterns in the area, the use of such water for drinking purposes should be discouraged. If it is necessary to use such water for drinking, residents should be advised to clean their cisterns regularly and to use the appropriate amount of chlorine.

773. So that the effects of fluorides ingested by the children of the area in 1965 and 1966 may be assessed, a dental survey of them should be conducted in 1970. The results of such a precautionary survey should be compared with the results of the 1968 survey, as recorded in this report.

774. An adequate and realistic method of assessing damage to livestock by pollutants should be available to veterinarians. This could be prepared by the University of Guelph and the Ontario Department of Agriculture and Food.

775. Crop losses should only be assessed on the basis of examination of standing crops. Procedures and criteria for assessing such damage could be prepared by the University of Guelph and the Ontario Department of Agriculture and Food.

776. The Ontario Department of Agriculture and Food or the University of Guelph should conduct field tests—as opposed to greenhouse tests—to determine susceptibility of different varieties of crops and vegetables to various air-borne pollutants. Such tests, if the fluoride emissions continue in the Port Maitland area, should be conducted there; such tests would help in determining the maximum concentrations of air-borne pollutants permissible in an area.

777. Consideration should be given to revising the *Canada Feeds Act* with respect to fluorides; present standards of fluoride in feed and mineral mixes, under certain circumstances, could produce a problem in livestock.

778. Cognizance should be taken of the unique position of the University of Guelph, through its Agriculture and Veterinary Science personnel, in present and future pollution control as related to crops, vegetation, soils, farm management, economics, plant sciences, animal sciences, etc. The support and utilization of such a group of scientists is indeed a must.

779. Until the pollution problem in the Port Maitland area has been finally solved, and we see no reason for delay, the full-time farmers and the part-time farmers in the area should be encouraged to modify somewhat their existing farm practices and farm management. Successful farming is possible in Florida and in the United Kingdom under conditions similar to those in the Port Maitland area in 1967.

780. We suggest that there should be a "let the hair down" meeting of all of those persons who were involved in this problem from the various branches of government. Some questions need answers. For example, does a Medical Officer of Health or an Agricultural Representative have a positive role to play in such a problem? Should they collect various samples and, if so, do they know the laboratory to which they should be sent? Who is the coordinator of the investigating team? How many laboratories should there be in the province conducting the same tests? Suffice it to say that fluoride determinations were being carried out in more than one of several government laboratories in the province. What procedure has been evolved to solve the problems of communication encountered in the Port Maitland investigations? A great deal can be learned through such a review; new or modified procedures and practices could be agreed upon. In other words, this *ad hoc* study of an area pollution problem should be used as a basis of discussion of pollution control in the province.

781. In addition to the above recommendations, the Committee of Inquiry makes a further major suggestion to the Government of the Province of Ontario. This is presented separately in the following chapter.

CHAPTER XIV

Basic Recommendations to the Government of Ontario

782. It is accurate to say that this study of the pollution problem in the Port Maitland area has been, in effect, an *ad hoc* study of industrial pollution in this province. It has shown the necessity of having a completely co-ordinated effort focused on the several aspects of what might superficially appear to be separate and distinct problems within the total area of concern. *The Air Pollution Control Act, 1967,* is a beginning in the provision of much needed co-ordination and co-operation.

783. As a result of inefficient or ineffective industrial process planning, one company to a major extent (and another company to a much lesser extent) did not satisfactorily control its air emissions from several parts of its plant, or from its waste-area lagoons, with the result that damage was caused to some crops and to some livestock. Such damage from air pollution was confined primarily to an area readily defined, and occurred over a period of several years.

784. There were many facets to this *ad hoc* study—the chief ones being the effects of the pollutants on:

(a) human beings: This facet was of critical concern. It revealed a potential human health problem and was especially significant to the medical and dental professions, the many specialists within those professions, and the Department of Health.

(b) cereal, forage, vegetable and fruit crops: The damage and the potential damage of pollutants on such crops was of the

utmost economic and agricultural concern. Such aspects then made this pollution problem one of special importance to the plant physiologist, the horticulturist, the agronomist, the agricultural economist, and other specialists in agricultural science, and to those in the Department of Agriculture and Food.

(c) cattle, sheep, hogs, poultry, and bees: The rearing of livestock in a polluted area was of great individual and general agricultural concern. These considerations are essentially in the domain of the veterinary scientist, clinician, pathologist, toxicologist, dental scientist, endocrinologist, and nutritionist, and again are of specific concern to the Department of Agriculture and Food.

(d) drinking water: Whether drinking water comes from deep wells, shallow wells, cisterns, or streams, it is a practical and important factor, not only in the human health field, but also in the health of livestock, where relatively large quantities of water are ingested. This is partially the concern of human health personnel and the Department of Health, and partially the concern of the Ontario Water Resources Commission.

785. When we attempt to translate some of the findings of this study into the larger sphere, other specific concerns appear. Some of them are listed here:

(a) The health of the workers in an industrial plant is a responsibility of the Department of Labour. Yet, when these same persons are in their homes in a polluted area they, like their families, are the responsibility of the Department of Health.

(b) If the pollutant is sulphur dioxide which emanates from an industrial plant, part of the responsibility lies with the Department of Mines.

(c) One of the two industrial plants in the area manufactures chemicals from an ore (marmatite) and, therefore, it is listed as a metallurgical plant and, as such, comes under the Department of Mines. Three hundred yards away, another plant which manufacturers chemicals from phosphate rock which, like marmatite, is dug from the earth, is not considered to be a metallurgical plant, and, therefore, comes under the Department of Health.

(d) Effluents from the two plants, relative to pollutants, is the concern of the OWRC, but the safety of well water, which could have as its original source seepage from the polluted stream (OWRC), is a responsibility of the Department of Health.

786. Perhaps the point has been made—the Departments of Mines, of Labour, of Lands and Forests, of Agriculture and Food, and of Health, and the OWRC, and several divisions within some of these Departments and agencies, were all involved at one time or another in the pollution problem in the Port Maitland area. This problem was known to exist in 1962. The OWRC has been involved with one of the companies since 1960. The "pollution problem" was still in existence at the conclusion of the hearings of this Committee in March, 1968.

787. Why was this problem not resolved sooner? The answers to this pertinent question are to be found throughout this report. But, as was mentioned in the report, HINDSIGHT IS BETTER THAN FORESIGHT. As a result of this study, we now have some of that HINDSIGHT to apply to the problems of the future.

788. Although some individuals in the Port Maitland area sustained damage to livestock and crops, nevertheless and fortunately, when looked at in the total provincial context, the damage was confined to a relatively small area. The damage was sufficient, however, to bring home to the Committee the necessity for the Provincial Government to initiate a study, in order to determine what kind of organization is required and best suited to make "pollution control" an effective and integrated reality.

789. It is with some reluctance that the Committee, at this stage in its report, does not now list or discuss some specific proposals relative to the control of pollution and the effects on humans, livestock, vegetation, wildlife, etc. May we point out that the problems of organization, of policy, of terms of reference, of teaching, of research, of laboratories, of people, of responsibilities, of communications, of jurisdictions, etc., are very complex. The study of such matters, urgent as they are, was not one of the tasks assigned to this Committee of Inquiry. It would be unwarranted and perhaps prejudicial to a thorough review of these considerations if we were to present here what, of necessity, would be superficial comments. We have learned a lot about part of the problem; the other parts deserve equal study.

790. Pollution is a natural and ingrained fact of life. Pollution control is yet in its infancy. We suggest that the government adopt an even greater sense of urgency in developing "anti-pollution" policies and in establishing the organization to implement them. We feel that a study of this extremely complex problem is of the utmost importance in the immediate future. Any changes which might be made in organization, in order that pollution control may be more effective, should be welcomed, even if recommendations emanating from such a study include the establishment of a Department of Environmental Health or a Department of Pollution Control.

Appendices

APPENDIX I

THE PUBLIC INQUIRIES ACT

ONTARIO COMMITTEE OF INQUIRY

"On allegations concerning pollution in the Townships of Dunn, Moulton and Sherbrooke"

NOTICE OF PUBLIC HEARINGS

On November 6, 1967, The Province of Ontario, through an Order-in-Council, established a Commission "to inquire into and report upon the pollution of air, soil and water in the Townships of Dunn, Moulton and Sherbrooke in the County of Haldimand and its effect upon human health, livestock, agricultural and horticultural crops, soil productivity and economic factors within the said areas."

In order that interested organizations, associations and individuals may have an opportunity to express their interest and concern in these matters, the Committee will hold public hearings at the Court House in Cayuga on January 22, 23, 24 and 26, as well as in Toronto. Any interested party or individual who wishes to present a brief or to make representation in person should contact the Committee in writing before January 12, 1968.

Notification of time and place of Public Hearings will be published in the near future.

FOR INFORMATION, PLEASE CONTACT—

M. Weissengruber,
Secretary, Pollution Committee,
1 St. Clair Avenue W., Toronto 7, Ont.

COPY OF NOTICE

1. Toronto Globe & Mail	December 23 and 25, 1967.
2. Hamilton Spectator	December 28 and 29, 1967.
3. Dunnville Chronicle	January 4, 1968.
4. Cayuga Haldimand Advocate	January 4, 1968.
5. Welland-Port Colborne Advocate	December 28 and 29, 1967.
6. St. Catharines Standard	December 28 and 29, 1967.
7. Niagara Falls Review	January 4 and 5, 1968.
8. Hagersville Press	January 4, 1968.

APPENDIX II

12th January, 1968.

FOR IMMEDIATE RELEASE

Further to a press release dated January 12, 1968, the Committee of Inquiry concerning Pollution in the Townships of Dunn, Moulton and Sherbrooke, wishes to announce that public hearings will begin at the Cayuga County Court House on January 22, at 10:00 a.m. Any interested party is invited to attend. The hearings will continue on January 23, 24 and 26. The members of the Committee are Dr. G. E. Hall, Orillia; Dr. W. C. Winegard, Guelph, and Mr. A. McKinney, Brampton.

Counsel for the Committee is Mr. R. A. Gordon, St. Catharines.

Dates for additional hearings will be published when such dates are established.

M. Weissengruber,
Secretary.

APPENDIX III

Report of Investigation No. X-68232

IDENTIFICATION OF THREE DUST SAMPLES

Ontario Department of Health,
Environmental Health Branch.

Three dust samples, received via the Air Pollution Laboratory of the Ontario Department of Health, have been examined by x-ray diffraction and x-ray spectrographic methods in order to identify the major crystalline components present.

Sample Identification

"Car Hood" refers to a sample of powder received in an envelope labelled "Dust swept from hood of car 10:00 a.m. 3-20-8." The powder was sieved through a 100 mesh screen in order to remove coarse wood and fibre particles. Only the fine fraction was analyzed.

"Frosted Bulb" refers to the surface deposit found on a frosted glass light bulb. This deposit forms a fairly heavy coating covering most of the glass envelope.

"Clear Bulb" refers to the surface deposit found on a clear glass light bulb. This deposit was fairly heavy on one side of the bulb only. Other areas of the bulb showed only a very thin deposit.

Results

Semi-quantitative x-ray spectrographic scans were done on each sample to assist with the interpretation of the x-ray diffraction patterns. The range of elements scanned included all elements with atomic number greater than 13. The elements detected are listed below.

	Car Hood	*Frosted Bulb*	*Clear Bulb*
Major Elements:	calcium	calcium	calcium
	sulphur	sulphur	sulphur
	silicon	lead	

Medium Elements:	potassium	potassium	potassium
	zinc	zinc	zinc
	phosphorus	phosphorus	phosphorus
	aluminum	aluminum	aluminum
	titanium	titanium	
		silicon	silicon
		cadmium	
Minor Elements:	iron	iron	iron
	nickel	nickel	nickel
	copper	copper	copper
	strontium		strontium
	lead		lead
	cadmium		cadmium
			titanium

The x-ray diffraction analyses gave the following identification of the major crystalline components present in each sample.

Car Hood			
— gypsum,	$CaSO_4.2H_2O$,	approximately	75%
— quartz,	$Si0_2$	"	8%
— zinc sulphide,	βZnS	"	3%
— calcite,	$CaCO_3$	"	3%
— talc,	(complex silicate)	"	3%
— feldspar,	(complex silicate)	"	3%
Frosted Bulb			
— gypsum,	$CaSO_4.2H_2O$,	approximately	25%
— lead sulphate,	$PbSO_4$	"	25%
— lead hydroxyapatite,	$Pb_5(PO_4)_3OH$	"	25%
— zinc sulphide,	βZnS	"	5%
Clear Bulb			
— gypsum,	$CaSO_4.2H_2O$,	approximately	50%
— calcium sulphate,	$CaSO_4$	"	25%
— zinc sulphide,	βZnS	"	10%
— quartz,	SiO_2	"	2%

Experimental Details

The "Car Hood" sample was passed through a 100 mesh sieve to remove coarse foreign particles such as wood and fibres. The fine fraction was then crushed to a fine powder before analysis. Samples were removed from both bulbs by scraping with a sharp stainless steel knife.

For the spectrographic scan, equal amounts of sample were used in each case, in order to permit a comparison between samples. Each spectrographic scan was done in two parts, one with a scintillation detector and LiF crystal for the heavy elements, the other with a flow counter and PET crystal in vacuum for the light elements.

Initially, x-ray diffraction patterns were obtained using the powder camera method. However the complexity of the patterns did not permit a satisfactory identification of the components by this method. Therefore new x-ray diffraction patterns were obtained with a recording diffractometer using copper K_α radiation and a curved-crystal monochromator. Interpretation of the diffraction patterns was based on reference data in A.S.T.M. Powder Diffraction File.

U. M. Martius, Ph.D.,
Consultant,
Department of Engineering
and Metallurgy.

E. Niskanen, P.Eng.,
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APPENDIX IV

Quantitative Relationship Between Natural Fluoride Concentration in the Common Water Supply and Severity of Enamel Mottling

		Per Cent Distribution of Mottling Severity				
Community	*Water Fluoride p.p.m.*	*Normal Question-able*	*Very Mild*	*Mild*	*Mode-rate*	*Severe*
Michigan City, Ind.	.1	100	0	0	0	0
Quincy, Ill.	.1	99.7	0.3	0	0	0
Elkhart, Ind.	.1	99.6	0.4	0	0	0
Middletown, Ohio	.2	98.9	1.1	0	0	0
Portsmouth, Ohio	.1	98.7	1.3	0	0	0
Zanesville, Ohio	.2	98.5	1.5	0	0	0
Lima, Ohio	.3	97.8	2.2	0	0	0
Marion, Ohio	.4	93.7	5.3	0.8	0	0
Pueblo, Colo.	.6	93.5	6.2	0.3	0	0
Kewanee, Ill.	.9	87.8	10.6	1.6	0	0
Aurora, Ill.	1.2	85.0	13.9	1.1	0	0
Joliet, Ill.	1.3	74.7	22.2	3.1	0	0
East Moline, Ill.	1.2-1.5	68.4	29.6	2.0	0	0
Maywood, Ill.	1.2-1.5	66.7	29.2	4.1	0	0
Elmhurst, Ill.	1.8	60.0	30.0	8.8	1.2	0
Galesburg, Ill.	1.9	52.4	40.3	6.2	1.1	0
Clovis, N.M.	2.2	27.9	21.8	36.9	12.3	1.1
Colorado Springs, Colo.	2.6	26.2	42.1	21.3	8.9	1.5
Plainview, Texas	2.9	12.4	34.0	26.8	23.7	3.1
Amarillo, Texas	3.9	9.7	15.2	28.0	33.9	13.2
Lubbock, Texas	4.4	2.0	12.2	21.7	46.0	17.9
Post, Texas	5.7	0	0	10.5	50.0	39.5

Source:

U.S. Public Health Reports 52, 1249-1264, (1937).

U.S. Public Health Reports 56, 761- 791, (1941).

U.S. Public Health Reports 57, 1155-1179, (1942).

APPENDIX V

Classification of Abnormalities of Enamel Related to Excessive Fluoride Intake

Classification	*Criteria of Classification*
NORMAL	Normal—no evidence of enamel abnormalities. Questionable—a) areas of enamel hypoplasia or hypocalcification due to exanthematous disease or nutritional disturbances. Such areas are readily distinguished from fluoride-induced abnormality by virtue of their sharp demarcation, their invisibilty when viewed in oblique light, and their predilection for the lower incisor region. b) white streaks or patches which are so faint that they are difficult to locate on re-examination even by the initial examiner.
VERY MILD	Small opaque paper-white areas, scattered irregularly or streaked over the tooth surface. Predilection for labial and buccal surfaces and involving less than 25 per cent of the surfaces of affected teeth. Small pitted white areas are frequently found on the summits of the cusps. No brown stain is present in mottled enamel of this classification.
MILD	The white, opaque areas involve more than 25 per cent of the tooth surface. Faint brown stains are sometimes apparent, generally on the upper incisors.
MODERATE	Tooth form is normal, but opaque; white enamel generally covers all tooth surfaces. Minute pitting is often present, generally on labial and buccal surfaces. Brown stain of the enamel is a frequent, disfiguring complication but one which is apparently related to some secondary factor (in the water?). The pitting (hypoplasia) is frequent and is generally observed on all tooth surfaces. In extreme cases the pits are confluent and so extensive as to affect the tooth form. Stains are widespread and range in colour from chocolate brown to almost black in some cases.

APPENDIX VI

INCIDENCE AND SEVERITY OF MOTTLED ENAMEL IN CHILDREN OF THE PORT MAITLAND AREA

Sub-group Comparison		*Group I Teeth*						*Group II Teeth*						*Groups I and II Teeth*					
		*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*	*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*	*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*
All Groups		119	29	2	1	0	151	51	12	1	0	0	64	170	41	3	1	0	215
Distance from Pt. Maitland (miles)	0.5-1.9	29	13	2	1	0	45	9	8	0	0	0	17	38	21	2	1	0	62
	2.0-2.9	28	7	0	0	0	35	14	3	0	0	0	17	42	10	0	0	0	52
	3.0-3.9	33	4	0	0	0	37	11	0	0	0	0	11	44	4	0	0	0	48
	4.0-6.9	29	5	0	0	0	34	17	1	1	0	0	19	46	6	1	0	0	53
Period of Tooth Group Development	1957-1961	42	15	1	0	0	58	10	3	0	0	0	13	52	18	1	0	0	71
	1958-64	49	9	1	1	0	60	41	9	1	0	0	51	90	18	2	1	0	111
	1961-65	28	5	0	0	0	33	0	0	0	0	0	0	28	5	0	0	0	33
Highest Severity Sub-groups																			
Sector A 0.5-1.9 m.		22	10	1	0	0	33	8	4	0	0	0	12	30	14	1	0	0	45
Sector B 0.5-1.9 m.		7	3	1	1	0	12	1	4	0	0	0	5	8	7	1	1	0	17

*The Normal category includes individuals with non-fluoride enamel abnormalities. Dean *et al* classified these individuals as questionable but noted that the defects were unrelated to fluoride mottling.

APPENDIX VII

PER CENT DISTRIBUTION OF MOTTLED ENAMEL SIGNS IN CHILDREN OF THE PORT MAITLAND AREA

Sub-group Comparisons	*Group I Teeth*						*Group II Teeth*						*Groups I and II Teeth*					
	*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*	*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*	*Normal**	*Very Mild*	*Mild*	*Moderate*	*Severe*	*Total*
All Groups	78.8	19.2	1.3	0.7	—	100.0	79.7	18.7	1.6	—	—	100.0	79.0	19.1	1.4	0.5	—	100.0
Distance from Pt. Maitland (miles) 0.5-1.9	64.5	28.9	4.4	2.2	—	100.0	53.0	47.0	—	—	—	100.0	61.4	33.8	3.2	1.6	—	100.0
2.0-2.9	80.0	20.0	—	—	—	100.0	82.3	17.7	—	—	—	100.0	80.8	19.2	—	—	—	100.0
3.0-3.9	89.2	10.8	—	—	—	100.0	100.0	—	—	—	—	100.0	91.7	8.3	—	—	—	100.0
4.0-6.9	85.4	14.6	—	—	—	100.0	89.4	5.3	5.3	—	—	100.0	86.8	11.3	1.9	—	—	100.0
Period of Tooth Group Development 1957-1961	72.5	27.5	—	—	—	100.0	76.9	23.1	—	—	—	100.0	73.1	25.5	1.4	—	—	100.0
1958-64	81.6	15.0	1.7	1.7	—	100.0	80.3	17.7	2.0	—	—	100.0	81.1	16.2	1.8	0.9	—	100.0
1961-65	85.0	15.0	—	—	—	100.0	—	—	—	—	—	—	84.9	15.1	—	—	—	100.0
Highest Severity Sub-groups																		
Sector A 0.5-1.9 m.	66.7	30.3	3.0	—	—	100.0	66.7	33.3	—	—	—	100.0	66.7	31.1	2.2	—	—	100.0
Sector B 0.5-1.9 m.	58.4	25.0	8.3	8.3	—	100.0	20.0	80.0	—	—	—	100.0	43.8	43.8	6.2	6.2	—	100.0

*The Normal category includes individuals with non-fluoride enamel abnormalities. Dean *et al* classified these individuals as questionable but noted that the defects were unrelated to fluoride mottling.

APPENDIX VIII

Electric Reduction Company of Canada, Ltd.
155 Etobicoke Drive Toronto (Islington) Ontario
Telephone 416-239-7111
Cable address Eiredchem

A member of the Albright and Wilson Group
Agricultural Chemicals Division

Mr. ______________________

At the Public Inquiry, ERCO stated that its President would review the whole question of pollution in the Spring and would not hesitate to make any reduction in production that might seem desirable. The purpose of this letter is to advise you that this has been done.

We are now installing the new scrubber on the calcium phosphate plant and are completely satisfied that there can be no cause for complaint from this source. The same is true of the wet acid plant which has been equipped over the years with a series of scrubbers.

The only area of manufacture which still causes us concern, despite our best efforts, is the plant in which single and triple superphosphate is made. We are still working on the problem of pile emissions and have decided this year to stop production of these products during the growing season. We ask you to appreciate that this is an expensive decision for us. We shall naturally take expert advice as to the appropriate date of resuming production.

By taking these and other measures we are convinced that you will have no cause to complain that your crops are affected by fluorides during the 1968 season, but in the event that you have any cause for alarm we are suggesting the following procedure.

As you know, ERCO has appointed Mr. Kenneth Couvillon as its Agriculture Liaison Officer in the Port Maitland area. He in turn has independent experts at his disposal. If, therefore, during the season you at any time suspect that your crops are affected in any way whatsoever, we ask you to contact Mr. Couvillon immediately and to allow him to photograph the crop and take a sample of it.

He may ask for the crop to be inspected by our expert. In return we undertake to make available to you the results of any tests that are made. If these tests should indicate fluoride then we undertake to negotiate an immediate compensation award directly with you. It is essential to this proposal that we have prompt notification of any suspected damage.

ERCO will not, for the year 1968, entertain any general arbitration as was carried out for the years 1965 and 1966 and as is presently being done for 1967. On the other hand, we make Mr. Couvillon available to you. His telephone number is 774-7476.

Your worries during the past years have been of great concern to us. We sincerely believe that these are all behind us and we extend our best wishes for a successful growing season in 1968.

Sincerely,

H. C. Hollands,
Senior Vice President.

APPENDIX IX

March 14, 1968.

Mr. L. Gosnell, Producer,
Canadian Broadcasting Corporation,
P.O. Box 500, Terminal "A",
2 Carlton Street,
Toronto, Ontario.

Dear Mr. Gosnell:

During the course of the study by the Committee of Inquiry into the alleged "pollution" in the Townships of Sherbrooke, Moulton and Dunn in the County of Haldimand, the Committee and its Counsel, together with Counsel for the Electric Reduction Company of Port Maitland, Ontario, by arrangement with Mr. Larry Gosnell, C.B.C., Toronto, viewed the film "Air of Death" which was presented as a C.B.C. special over Channel 6, Sunday, October 22nd, 1967, at 8:00 p.m.

Prior to viewing this particular film, copies of the script were obtained from Larry Gosnell, Producer

The Committee, interested in obtaining the facts and attempting to establish the truth in connection with this complex problem, as it relates solely to the above-mentioned townships, wishes to ask the C.B.C. several pertinent and relevant questions and would appreciate receiving appropriate and precise answers to such questions. These questions are listed in the attached questionnaire.

In view of the amount of research which was stated to have been done prior to the preparation of the film, the Committee would hope to have your completed reply by the 22nd of this month.

Yours very truly,

Max E. Weissengruber,
Secretary.

QUESTIONNAIRE RE CBC FILM "AIR OF DEATH"

1. (a) STATEMENT—made in Film—p. 1 of script.
"For the past six months we've been researching this problem."

(b) QUESTION—In view of the great complexity of the problem of air, water and soil pollution and comments made subsequently in the film, please list the names of your research advisers or consultants concerning the following disciplines: veterinarian, physician, agronomist, physiologist, toxicologist, horticulturist and industrial chemist or engineer.

2. (a) STATEMENT—made in film—p. 1 of script.
"You breathe sulphur dioxide which erodes stone."

(b) QUESTION—(1) list references which would show "that sulphur dioxide erodes stone."

(2) from your study of the literature, what are the accepted limits of tolerance of sulphur dioxide in the air, at the five-foot levels in the United Kingdom, the Ruhr area of West Germany, in the industrialized areas of the U.S.S.R. and in various centres in the United States?

3. (a) STATEMENT—in film—p. 1 of script.
"They [air pollution] cut a hole in your throat."

(b) QUESTION—Please provide clinical and/or pathological evidence for this statement.

4. (a) STATEMENT—in film—p. 1 of script.
"lung diseases as a whole are now the number one killer in Canada . . ."

(b) QUESTION—(1) Please provide supporting evidence and references.

(2) Please list major causes of deaths in Canada in the different population age groups.

5. (a) STATEMENT—in film—p. 1 of script.
"sulphur dioxide, which in the lungs forms sulphuric acid."

(b) QUESTION—Please provide supporting evidence and references.

6. (a) STATEMENT—in film—p. 22 of script.
" . . . from some of the richest farming land in the country."

(b) QUESTION—Please supply supporting evidence on the following suggested basis:

(i) a comparison of the average yields of grain and fodder in the Townships of Dunn, Moulton and Sherbrooke in particular, and of Haldimand County generally with the average yield of the same grain and fodder in the neighbouring counties of Welland and Lincoln and in the other Counties in South Western Ontario, using two consecutive census years data or the agricultural statistics of Ontario from 1955-1967.

(ii) the average value of farm land, per acre, in the Townships of Dunn, Moulton and Sherbrooke, over the same census periods, as compared to any three townships in any other county in Western Ontario.

(iii) a comparison of soil types, soil drainage, size of farm, etc.

7\. (a) STATEMENTS—in film—p. 22 of script.

(i) "dwarfed and shrivelled the grain"

(ii) "damaged everything that grew"

(b) QUESTION—(i) please provide evidence which would support the above two statements.

(ii) what concentrations of sulphur dioxide and of fluoride respectively at the five-foot level would be required to produce the above damage, which the above two statements claim?

8\. (a) STATEMENT—in film—p. 23 of script.
" . . . grain yields cut in half"

(b) (refer to question 6 (b) (i): please give evidence relative to the following grain crops: shelled corn, winter wheat, oats, barley, on a comparative basis, over a ten-year period and with yields for the same grain crops in another county of Southern Ontario over the same ten-year period.

9\. (a) STATEMENT—in film—p. 23 of script.
"Fluorosis—swollen joints, falling teeth, pain, until [the] cattle lie down and die—hundreds of them."

(b) QUESTIONS—(i) How many cattle were there on the farms in Dunn, Moulton and Sherbrooke Townships in 1955, 1961, 1965, and 1967? And in the years 1961, 1965, 1967—how many cattle had been diagnosed by a veterinarian as having fluorosis, and the degree of fluorosis.

(ii) please provide evidence of "falling teeth" and the number of cattle from which teeth had fallen out.

(iii) please provide evidence that "the cattle lie down and die."

(iv) to support the statement " . . . hundreds of them" please provide appropriate evidence in the way of numbers, farm location and ages and breed of cattle.

10. (a) REFERENCE STATEMENTS — in film — p. 23-24 of script by Mr. Joe Casina, Sr.

(b) QUESTION—In view of the evidence presented at the Hearings of the Committee of Inquiry by Dr. Marson, and Mr. Joe Casina, wherein he stated "In 1952, I had a heart attack" and on further questioning admitted having a second heart attack in 1964, were Dr. Marson and Dr. Green contacted or interviewed prior to October 22nd, 1967—and if not, why not?

11. (a) STATEMENT—in film by "Interviewer"—p. 28 of script. "when we have seen the crop damage that we know, I mean that can be recognized, as specific fluorine damage, —marks on the leaves, we've seen cattle that are crippled, with their teeth falling out . . ."

(b) QUESTION — (i) How did your research advisers describe leaf damage resulting from excess exposure to fluoride and how is such leaf damage differentiated from damage by sulphur dioxide, which is one of the pollutants from a plant in the area, from copper and other trace element deficiency damage and from damage resulting from one or more plant diseases or insects?

(ii) please provide evidence that such "marks on the leaves" were . . . "as specific fluorine damage."

12. (a) STATEMENT—in film—p. 31 of script.
"about the health issue at Dunnville"

(b) QUESTION—(i) What is the distance from the ERCO and Sherbrooke Metallurgical plants in Port Maitland to the Town of Dunnville?

(ii) What is the direction of the prevailing winds at Port Maitland?

(iii) What is the maximum fluoride concentration of air, water, soil, vegetation recorded in Dunnville during the past 8 years?

(iv) Why was Dunnville, by implication, associated with this problem?

13. (a) STATEMENT — in film — Mr. Larry Gosnell — p. 31 of script.

"What's going to happen to these people who have fluoride poisoning?"

(b) QUESTION—(i) What are the accepted signs and symptoms of fluoride poisoning?

(ii) Please give detailed evidence, which warranted the above statement.

(iii) Please provide the names and addresses of "these people who have fluoride poisoning."

14. (a) STATEMENT—in film—p. 36 in script.
"I've driven through Germany, the industrial heartland of Europe, and the air is clear. Russia has imposed the highest standards of purity in the world."

(b) QUESTION—Please list the allowable levels of sulphur dioxide, fluoride, carbon monoxide and particulate smoke at five feet, on ground levels, in the United Kingdom, the industrial areas of the U.S.S.R., the Ruhr area of West Germany, central Florida, and other relevant areas in the United States, together with permissible stack emission levels of pollutants and a comparison of those standards to actual day to day, week to week concentrations of the same pollutants in the same areas.

Your assistance in providing the Committee with the above requested information will be of great assistance in supplementing the information provided to the Committee through evidence, through surveys of the relevant scientific literature and through supporting documents prepared by the Committee's several Consultants.

APPENDIX X

CANADIAN BROADCASTING CORPORATION
SOCIETE RADIO-CANADA

P.O. Box 500
Toronto, Ontario
22 March 68

Mr. Max E. Weissengruber,
Secretary,
Committee of Inquiry,
1 St. Clair Avenue West,
Toronto 7, Ontario.

Dear Mr. Weissengruber:

This is in reply to your letter dated March 14th, addressed to Mr. Gosnell, Producer, and enclosing a lengthy questionnaire concerning the preparation of one of the Corporation's programs broadcast October 22nd, 1967.

We understand that the Commission's terms of reference, as quoted to us by the Commission's counsel in previous correspondence, are not to enquire into the preparation of the program but "to enquire and report upon the pollution of air, soil and water in the Townships of Dunn, Moulton and Sherbrooke in the County of Haldimand and its effects upon human health, livestock, agricultural and horticultural crops, soil productivity and economic factors within the said area."

Quite apart from the terms of reference actually given to the Commission, we do not believe that a Commission established by a provincial jurisdiction could be given authority to enquire into the preparation of a C.B.C. program. As you know, the C.B.C. has been established by Parliament for the purpose of operating a national broadcasting service and is accountable to Parliament for the conduct of its affairs and the discharge of its responsibilities.

Consequently, we do not consider that it would be in order for us to reply to the questions contained in your questionnaire.

Yours very truly,
Marce Munro
Acting General Manager,
Network Broadcasting,
(English)

APPENDIX XI

March 27, 1968.

Mr. M. Munro
Acting General Manager
Network Broadcasting (English)
Canadian Broadcasting Corporation
P.O. Box 500,
Toronto, Ontario.

Dear Mr. Munro:

This will acknowledge receipt of your letter of March 22nd in reply to Mr. Weissengruber's letter to Mr. Gosnell dated March 14th, 1968.

As Chairman of the Committee of Inquiry into the matter of pollution in the Townships of Dunn, Moulton and Sherbrooke in the County of Haldimand, I wish to assure you that the Commissioners are most anxious to obtain all the assistance possible to understand and to assess this most complex and significant problem, and subsequently to make realistic recommendations which will help prevent and to control comparable situations.

We recognize the great responsibility which the Canadian Broadcasting Corporation has exercised in presenting its programmes to the people and have noted with special interest the fact that before presenting the film "Air of Death" it conducted extensive research into the problem. That portion of the film dealing with pollution in the so-called Dunnville area contained results of your research which are of extremely great interest to the Commissioners, varying, as many of your results did, with some of the evidence obtained at the Public Hearings and with the reported scientific literature.

Citing a few examples of the divergence of your research results—which give the Commissioners great concern—may indicate the honest efforts which we are making in order to report fully and in an unbiased fashion the results and conclusions of all relevant research studies. I appreciate that you must have such evidence or the C.B.C. would not have made the statements which it did.

(a) "They (air pollution) cut a hole in your throat" (Page 1 of Script.)
We can find no evidence, relative either to man or livestock, to support this finding.

(b) (1) "Damage everything that grew" (Page 22 of Script.)
(2) "dwarfed and shrivelled the grain."
Again, no farmer or resident in the area gave such evidence, nor can we find substantiating evidence in the literature.

(c) " . . . cattle lie down and die—hundreds of them" (page 23 of script.)
We were not able to elicit any evidence at the Hearings which supported this claim. Evidence from major surveys and research studies in the United Kingdom and the United States likewise is contrary to the results of your research.

(d) "What's going to happen to these people who have fluoride poisoning?" (page 31 of script.)
On the basis of evidence of local physicians, of an area consulting physician and of extensive investigations, under hospitalized conditions in one of Canada's foremost teaching and research hospitals, none has been presented which would indicate that there are "people who have fluoride poisoning."

These few examples, which cause us some perplexity, should suffice to indicate that unless we receive substantiated contrary evidence, our report will of necessity be contradictory to many of the statements made by the C.B.C.

Naturally, therefore, it is most desirable that we solicit your assistance and benefit from the studies which your colleagues have made relevant to the problem in the Port Maitland area. It was for these reasons, and in a collaborative spirit, that the Committee asked Mr. Gosnell for his help through providing answers to the questions contained in the questionnaire submitted to him with the letter of March 14th.

Your co-operation will be of significant assistance in helping the Commissioners arrive at responsible decisions and in making their report an effective one. We would be most appreciative if the answers to the questions could be received by April 10th, 1968.

Yours very truly,
G. Edward Hall,
Chairman.

APPENDIX XII

LIST OF WITNESSES

Persons Appearing at Public Hearings

1. Dr. P. J. Lawther,
Director, Medical Research Council Unit for Air Pollution Research,
St. Bartholomew's Hospital, Medical College,
Charterhouse Square,
London, E.C. 1, England.

2. Dr. A. E. Martin,
Senior Medical Officer,
Ministry of Health,
Alexander House,
Elephant and Castle,
London, S.E. 1, England.

3. Dr. W. C. B. Mills,
Medical Officer of Health,
Townships of Sherbrooke and Moulton,
Dunnville, Ontario.

4. Dr. F. D. Rigg,
Physician,
Dunnville, Ontario.

5. Dr. C. B. Greene,
Physician,
Dunnville, Ontario.

6. Mr. D. Bogaerts,
Senior Pesticides Inspector,
Pesticides Control Service,
Ontario Department of Health,
Toronto, Ontario.

7. Mr. W. M. Warnick,
R.R. #2,
Lowbanks, Ontario.

8. Mr. P. G. DeRuiter,
R.R. #2,
Lowbanks, Ontario.

9. Mrs. S. Nie,
R.R. #2,
Lowbanks, Ontario.

10. Mrs. J. MacKeigan,
R.R. #6,
Dunnville, Ontario.

11. Mr. C. Kneider,
President,
Dunnville Chamber of Commerce,
Dunnville, Ontario.

12. Mrs. A. Frith,
Vice-President,
Dunnville Chamber of Commerce,
Dunnville, Ontario.

13. Dr. E. Mastromatteo,
Chief, Occupational Health Service,
Ontario Department of Health,
Toronto, Ontario.

14. Dr. V. Tidey,
Medical Officer,
Occupational Health Service,
Ontario Department of Health,
Toronto, Ontario.

15. Dr. F. G. Marson,
Physician,
Hamilton, Ontario.

16. Dr. K. J. R. Wightman,
Professor of Medicine,
Faculty of Medicine,
University of Toronto,
Toronto, Ontario.

17. Mr. F. Schofield,
Clerk-Treasurer,
Town of Dunnville,
Dunnville, Ontario.

18. Mr. A. Farr,
R.R. #2,
Lowbanks, Ontario.

19. Mr. J. Casina, Sr.,
R.R. #6,
Dunnville, Ontario.

20. Mr. J. Vanderbeek,
R.R. #6,
Dunnville, Ontario.

21. Dr. J. L. Sullivan,
Consultant, Atmospheric Pollution,
Department of National Health and Welfare,
Ottawa, Ontario.

22. Mr. L. Work,
Technical Superintendent,
Electric Reduction Company Ltd.,
Port Maitland, Ontario.

23. Mr. C. Duncan,
Waste Control Engineer,
Electric Reduction Company Ltd.,
Port Maitland, Ontario.

24. Mr. D. P. Caplice,
Director, Industrial Waste Division,
Ontario Water Resources Commission,
Toronto, Ontario.

25. Mr. R. Neider,
Plant Superintendent,
Sherbrooke Metallurgical Company Ltd.,
Port Maitland, Ontario.

26. Mr. W. B. Drowley,
Chief, Air Pollution Control Service,
Ontario Department of Health,
Toronto, Ontario.

27. Mr. T. McAlonan,
R.R. #6,
Dunnville, Ontario.

28. Mr. R. Boorsma,
R.R. #2,
Lowbanks, Ontario.

29. Mr. A. Bruinsma,
R.R. #6,
Dunnville, Ontario.

30. Mr. L. McIntee,
R.R. #6,
Dunnville, Ontario.

31. Mr. K. Wright,
R.R. #6,
Dunnville, Ontario.

32. Dr. S. J. Slinger,
Professor and Chairman,
Department of Nutrition,
University of Guelph,
Guelph, Ontario.

33. Dr. R. Willoughby,
Professor of Veterinary Science, and Head of Medicine Section,
Ontario Veterinary College,
University of Guelph,
Guelph, Ontario.

34. Mr. E. Deamude,
R.R. #6,
Dunnville, Ontario.

35. Mr. R. Elkow,
R.R. #6,
Dunnville, Ontario.

36. Dr. J. Suttie,
Associate Professor,
Department of Biochemistry,
University of Wisconsin,
Madison, Wisconsin, U.S.A.

37. Mr. E. Puhl,
R.R. #2,
Lowbanks, Ontario.

38. Professor L. Webber,
Department of Soil Science,
University of Guelph,
Guelph, Ontario.

39. Dr. J. Tanner,
Associate Professor,
Crop Science Department,
University of Guelph,
Guelph, Ontario.

40. Dr. M. Brown,
Associate Professor,
Agricultural Meteorology,
Department of Soil Science,
University of Guelph,
Guelph, Ontario.

41. Professor J. Clark,
Department of Agricultural Economics,
University of Guelph,
Guelph, Ontario.

42. Mr. K. K. Huffstutler,
Engineer-in-Charge,
Environmental Health Facilities,
Florida State Board of Health,
Winterhaven, Florida, U.S.A.

43. Mr. R. Paisley,
R.R. #6,
Dunnville, Ontario.

44. Mr. J. Ostirc,
R.R. #5,
Port Maitland, Ontario.

45. Mr. D. Dick,
Dunnville, Ontario.

46. Mr. J. Carruthers,
R.R. #6,
Dunnville, Ontario.

47. Mr. H. Rittenhouse,
R.R. #6,
Dunnville, Ontario.

48. Mr. E. Harvey,
R.R. #2,
Lowbanks, Ontario.

49. Mr. L. Niece,
R.R. #2,
Lowbanks, Ontario.

50. Mr. P. Shelestowsky,
R.R. #2,
Lowbanks, Ontario.

51. Mr. A. Hoto,
R.R. #2,
Lowbanks, Ontario.

52. Mr. P. Purych,
R.R. #2,
Lowbanks, Ontario.

53. Professor J. Clark
(Second appearance).

54. Mr. E. McQuillan,
Clerk-Treasurer,
Township of Moulton,
Ontario.

55. Mr. R. Elkow,
(Second appearance).

56. Mr. A. Karl,
R.R. #6,
Dunnville, Ontario.

57. Mr. J. Casina, Sr.,
(Second appearance).

58. Dr. F. C. Nelson,
Head, Regional Veterinary Science Laboratory,
Ontario Department of Agriculture and Food,
Guelph, Ontario.

59. Mr. W. B. Drowley,
(Second appearance).

60. Dr. E. Mastromatteo,
(Second appearance).

61. Mr. G. Dickout,
R.R. #2,
Lowbanks, Ontario.

62. Mr. E. Siddall,
R.R. #7,
Dunnville, Ontario.

63. Mr. G. Martin,
R.R. #2,
Lowbanks, Ontario.

64. Mr. W. Siddall,
R.R. #2,
Lowbanks, Ontario.

65. Mr. D. Middleton,
Secretary,
Ontario Federation of Agriculture,
Toronto, Ontario.

66. Dr. J. Tanner,
(Second appearance).

67. Mr. C. Duncan,
(Second appearance).

APPENDIX XIII

SUMMARY OF ARBITRATION AWARDS FOR THE YEARS 1965, 1966, AND 1967

Name	Year	Livestock	Field Crops & Horticulture	Total
D. Boorsma	Award 1965	16,510.75	3,525.39	20,036.14
	Award 1966	2,630.00	2,866.97	5,496.97
	Award 1967	600.00	5,174.75	5,774.75
				31,307.86
J. Carruthers	Award 1965	1,720.00	4,135.68	5,855.68
	Award 1966	3,240.00	3,266.39	6,506.39
	Award 1967		2,564.00	2,564.00
				14,926.07
Elkow Bros.	Award 1965	6,289.05	3,614.00	9,903.05
	Award 1966	9,320.00	9,424.05	18,744.05
	Award 1967			
				28,647.10
E. Deamude	Award 1965	825.00	1,215.89	2,040.89
	Award 1966		585.93	585.93
	Award 1967		1,951.04	1,951.04
				4,577.86
P. Krickhan	Award 1965	1,107.00	1,752.70	2,859.70
	Award 1966	140.00	1,366.20	1,506.20
	Award 1967		2,445.80	2,445.80
				6,811.70
T. McAlonan	Award 1965	6,250.00	2,188.81	8,438.81
	Award 1966		2,441.06	2,441.06
	Award 1967	4,785.00	3,483.00	8,268.00
				19,147.87
P. Shelestowsky	Award 1965	3,851.78	713.19	4,564.97
	Award 1966		2,408.22	2,408.22
	Award 1967		2,255.20	2,255.20
				9,228.39

Name	*Year*	*Livestock*	*Field Crops & Horticulture*	*Total*
J. Vanderbeek	Award 1965	6,396.56	3,264.20	9,660.76
	Award 1966		5,734.24	5,734.24
	Award 1967		2,275.00	2,275.00
				17,670.00
J. Casina Sr. & Jr.	Award 1965		2,434.64	2,434.64
	Award 1966	235.00	6,454.79	6,689.79
	Award 1967		9,863.83	9,863.83
				18,988.26
K. Wright	Award 1965	2,578.50	2,674.51	5,253.01
	Award 1966	1,960.00	2,968.01	4,928.01
	Award 1967		3,744.05	3,744.05
				13,925.07
T. Zynomirski	Award 1965	2,067.00	1,176.39	3,243.39
	Award 1966	333.00	2,243.84	2,576.84
	Award 1967		1,686.00	1,686.00
				7,506.23
L. McIntee	Award 1965		4,961.00	4,961.00
	Award 1966	3,640.00	5,824.40	9,464.40
	Award 1967		6,579.00	6,579.00
				21,004.40
Harvey Bros.	Award 1965			
	Award 1966	3,095.00		3,095.00
	Award 1967			
				3,095.00
F. Jonke	Award 1965			
	Award 1966	6,945.00	582.82	7,527.82
	Award 1967		382.00	382.00
				7,909.82
Niece Bros.	Award 1965			
	Award 1966	1,280.00		1,280.00
	Award 1967		320.00	320.00
				1,600.00

Name	Year	Livestock	Field Crops & Horticulture	Total
R. Paisley	Award 1965			
	Award 1966	6,995.00	1,308.80	8,303.80
	Award 1967		315.00	315.00
				8,618.80
E. Puhl	Award 1965			
	Award 1966	2,285.00	1,161.33	3,446.33
	Award 1967	148.50	2,799.50	2,948.00
				6,394.33
P. Purych	Award 1965		90.50	90.50
	Award 1966	1,175.00		1,175.00
	Award 1967		218.00	218.00
				1,483.50
M. Pyle	Award 1965			
	Award 1966	1,605.00	204.00	1,809.00
	Award 1967			
				1,809.00
H. Rittenhouse	Award 1965			
	Award 1966	2,750.00		2,750.00
	Award 1967			
				2,750.00
A. Karl	Award 1965		3,626.68	3,626.68
	Award 1966		3,095.28	3,095.28
	Award 1967		4,747.00	4,747.00
				11,468.96
J. Gibson	Award 1965		1,117.53	1,117.53
	Award 1966		1,410.30	1,410.30
	Award 1967		347.50	347.50
				2,875.33
L. Anderson	Award 1965		80.50	80.50
	Award 1966		1,104.80	1,104.80
	Award 1967	900.00	999.00	1,899.00
				3,084.30

Name		Year	Livestock	Field Crops & Horticulture	Total
M. Duda		Award 1965			
		Award 1966		2,600.10	2,600.10
		Award 1967		1,475.10	1,475.10
					4,075.20
J. Smith		Award 1965		1,219.89	1,219.89
		Award 1966		1,487.06	1,487.06
		Award 1967		1,831.34	1,831.34
					4,538.29
Mrs. E. Smith		Award 1965			
		Award 1966		1,547.58	1,547.58
		Award 1967		1,134.00	1,134.00
					2,681.58
Mr. & Mrs. Wm. Siddall		Award 1965		177.70	177.70
		Award 1966		605.00	605.00
		Award 1967		691.88	691.88
					1,474.58
Mrs. V. Farr		Award 1965			
		Award 1966		513.98	513.98
	(Bees)	Award 1967	*48.00*	1,367.25	1,415.25
					1,929.23
J. Cybulka		Award 1965			
		Award 1966		472.68	472.68
		Award 1967		457.25	457.25
					929.93
A. Hoto		Award 1965		236.65	236.65
		Award 1966		169.34	169.34
		Award 1967			
					405.99
J. Kinaschuk		Award 1965			
		Award 1966			
		Award 1967		1,505.00	1,505.00
					1,505.00

Name	Year	Livestock	Field Crops & Horticulture	Total
L. Richardson	Award 1965			
	Award 1966		427.06	427.06
	Award 1967	526.50	675.00	1,201.50
				1,628.56
Mr. & Mrs. Wayne Siddall	Award 1965		40.00	40.00
	Award 1966			
	Award 1967		219.50	219.50
				259.50
R. Hall	Award 1965		5.00	5.00
	Award 1966			
	Award 1967			
				5.00
E. Dickout	Award 1965			
	Award 1966		275.50	275.50
	Award 1967		133.00	133.00
				408.50
Bruce Siddall	Award 1965		25.00	25.00
	Award 1966		10.00	10.00
	Award 1967			
				35.00
Mr. & Mrs. G. Dickout	Award 1965			
	Award 1966		290.72	290.72
	Award 1967		782.16	782.16
				1,072.88
E. Anderson	Award 1965			
	Award 1966			
	Award 1967		930.50	930.50
				930.50
B. Girling	Award 1965			
	Award 1966			
	Award 1967		220.00	220.00
				220.00

Name	Year	Livestock	Field Crops & Horticulture	Total
D. MacArthur	Award 1965		10.00	10.00
	Award 1966		15.00	15.00
	Award 1967			
				25.00
F. Ross	Award 1965		114.50	114.50
	Award 1966		9.65	9.65
	Award 1967			
				124.15
E. Siddall	Award 1965		30.00	30.00
	Award 1966			
	Award 1967			
				30.00
D. R. MacKeigan	Award 1965			
	Award 1966		93.34	93.34
	Award 1967			
				93.34
Mrs. S. Nie	Award 1965		10.00	10.00
	Award 1966		117.35	117.35
	Award 1967			
				127.35
C. McHugh	Award 1965		5.00	5.00
	Award 1966		47.00	47.00
	Award 1967			
				52.00
A. Lindsay	Award 1965		9.20	9.20
	Award 1966			
	Award 1967			
				9.20
H. Pyle	Award 1965			
	Award 1966		403.20	403.20
	Award 1967			
				403.20

Name	*Year*	*Livestock*	*Field Crops & Horticulture*	*Total*
M. Pyle	Award 1965			
	Award 1966		117.00	117.00
	Award 1967			
				117.00
C. Cook	Award 1965		15.00	15.00
	Award 1966			
	Award 1967			
				15.00
Mr. D. Siddall	Award 1965			
	Award 1966		35.00	35.00
	Award 1967			
				35.00
Mrs. L. Siddall	Award 1965		38.00	38.00
	Award 1966		12.30	12.30
	Award 1967			
				50.30
A. Clark	Award 1965		40.75	40.75
	Award 1966		205.00	205.00
	Award 1967			
				245.75
Miss E. Vaughan	Award 1965			
	Award 1966		55.00	55.00
	Award 1967			
				55.00
G. Clark	Award 1965		25.00	25.00
	Award 1966		247.45	247.45
	Award 1967			
				272.45
V. King	Award 1965			
	Award 1966		386.00	386.00
	Award 1967			
				386.00

Name	*Year*	*Livestock*	*Field Crops & Horticulture*	*Total*
W. Clark	Award 1965		20.00	20.00
	Award 1966			
	Award 1967			
				20.00
Harry Downey	Award 1965			
	Award 1966			
	Award 1967	600.00	935.00	1,535.00
				1,535.00
Joseph V. Keitz	Award 1965			
	Award 1966			
	Award 1967		718.70	718.70
				718.70

ARBITRATION AWARD TOTALS

Year	*Livestock*	*Field Crops & Horticulture*	*Total*
Award 1965	47,595.64	38,593.30	86,188.94
Award 1966	47,628.00	64,593.74	112,221.74
Award 1967	7,608.00	65,225.35	72,833.35
	102,831.64	168,412.39	271,244.03

The above figures are the actual amounts paid to and received by the individuals listed, as of September 25, 1968.

APPENDIX XIV

Re: DR. G. L. WALDBOTT, DETROIT

As a result of the CBC film "Air of Death," the Committee was aware of the fact that Dr. G. L. Waldbott had taken an interest in the Port Maitland "pollution" problem. The Committee accordingly extended an invitation to Dr. Waldbott to appear before the Committee to give evidence relating to his findings as a result of the investigations carried out by him.

In view of the fact that Dr. Waldbott has already complained in a letter to the Press that he was not given an opportunity to appear, the Committee can only assume that he will repeat such complaints following the publication of this report.

The Committee, therefore, finds it necessary to state categorically that it was at all times during the hearings ready and willing to hear evidence from Dr. Waldbott and that the correspondence relative to his appearance before the Committee clearly establishes that fact.

The Committee cannot leave this matter without referring to the fact that Dr. Waldbott did prepare a brief with a covering letter dated April 17, 1968, 27 days after the hearings concluded, addressed to the Honourable Minister of Health, Dr. Matthew B. Dymond, but which was not mailed to the Minister of Health. It was in fact delivered to the Minister of Health by a member of the Ontario Legislature and the Minister of Health in turn relayed it to the Committee.

Despite the impropriety of the method used by Dr. Waldbott to make his views known and despite the fact that he saw fit not to submit himself for cross-examination on his brief, the Committee in an effort to do full justice to his views, which are well known in the medical world, not only considered the brief, but submitted it for analysis to Dr. Edmund Martin, Senior Medical Officer, Environmental Health Branch of the Ministry of Health in the United Kingdom, who had been retained as a Consultant by the Committee.

Dr. Martin submitted a complete review and report of Dr. Waldbott's brief, which has been studied by the Committee. The Committee rejects many of the statements made by Dr. Waldbott in his brief and accepts the testimony of the physicians and other scientists received in evidence and referred to or quoted in the Committee's report.

APPENDIX XV

LIST OF EXHIBITS

1. Copies of Orders-in-Council appointing Committee.
2. General outline of Committee's proposed activities.
3. Press release and distribution list.
4. Study entitled "Port Maitland Pollution Study."
5. Aerial photograph of area mentioned in Exhibit 4.
6. Private papers of the witness, Mr. W. Warnick.
7. Brief submitted by Dunnville Chamber of Commerce.
8. Report on vegetation sampling in 1965.
9. Article from *Toronto Daily Star,* "Dunnville fluoride too high."
10. Department of Health summary of fluoride results up to 1967.
11. Department of Health Laboratory Report on water fluoride content.
12. Analysis entitled "snow for fluoride" (content).
13. Report re "samples from Port Maitland."
14. Department of Health analyses for fluoride in urine and blood.
15. Medical reports on Messrs. Casina and Vanderbeek.
16. Copies of X-rays of Messrs. Casina and Vanderbeek.
17. Urinary analyses submitted in code.
18. Letters from Committee Secretary to Dr. Waldbott, and reply.
19. Brief from Mayor of Dunnville.
20. Pamphlet entitled "ERCO TODAY."
21. Diagram of ERCO's industrial processes.
22. Summary of Anti-pollution Capital Investment by ERCO.
23. Summary of OWRC pollution control activities, re ERCO.
24. Letter from Department of Lands and Forests to Department of Health.

25. (A and B) Basic reference maps for Port Maitland.

26. Summary of activities of the Air Pollution Control Service.

27. (A through G) Maps of Port Maitland area showing air monitoring data, crop, livestock damage, for years 1965 through 1967.

28. Article from the *Journal of the Air Pollution Control Association.*

29. Letter from D. Boorsma to Federal and Provincial Officers.

30. List of damage claims submitted.

31. Letter from Mr. W. B. Drowley to Mr. E. Deamude.

32. Table for assessing damage to teeth from fluoride.

33. Analysis of hay and cattle urine for fluoride.

34. Production figures from ERCO and Sherbrooke Metallurgical Co.

35. Letter from Mr. P. Hisem to Mr. L. Niece.

36. Statement filed by Mr. J. Casina.

37. Report on pollution abatement equipment—ERCO.

38. Report filed by Dr. F. C. Nelson.

39. Threshold Limit Values for various substances.

40. Piece of window glass.

41. Dust collected from car of Mr. G. Dickout.

42-A. Slightly discoloured light bulb.

42-B. Heavily discoloured light bulb.

43. Submission of the OFA to Ontario Law Reform Commission.

44. List of members of film selection committee, Council of Resource Ministers.

45. Letter from Mr. W. B. Drowley to ERCO.

APPENDIX XVI

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